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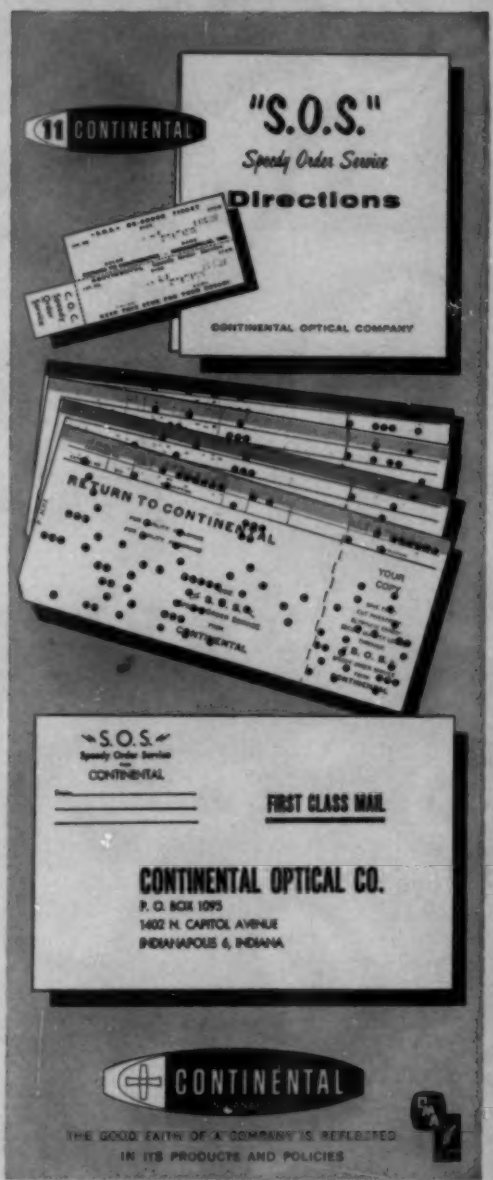

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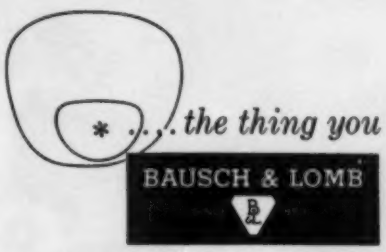

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WHY DOESN'T SHURON HAVE A "CAPTIVE" LABORATORY SYSTEM?

ROY MARKS
President, Shuron Optical Company

Practitioners often ask me and other Shuron people why our company doesn't maintain its own laboratories. Often rumors crop up to the effect that Shuron is opening a captive lab system, apparently started by those who feel that we should enter this part of the optical business.

The question comes up often enough so that I think it is important that we answer it for all . . . so there will be no mistaking our basic business philosophy.

First of all, Shuron has capital available through its parent company for such a venture. We have had, and I'm sure will have again, the opportunity either to purchase existing laboratories or to start new ones. So it is apparent that our reasons for not going into this business are not lack of money or opportunity.

The basic reason that we do not go into the laboratory business is that we feel the manufacturing function and the laboratory-distribution function operate best independently of each other. Only when they *are* independent do they offer the most in *service* and *product* to you.

If, for instance, Shuron were to serve this dual function, we would have conflicting interests within the same organization. Economics would dictate that we would have to be a manufacturer first, a lab-distributor second. Our thinking would be necessarily that of a manufacturer. Any laboratory-distribution network of ours would have conflicting functions, too. The laboratories would have to strive to serve you in the best possible way with a broad range of service and products. They would also have the conflicting responsibility of distributing the products of the manufacturing arm of the organization first—other products, second. The relative merits of the parent manufacturer's products could not be questioned by the captive laboratory. They would have to sell any and all products of the company with a partisan fervor.

We would certainly have to continue to sell to independent distributors. But we would be forced into the contradictory posi-

Report:

tion of being a supplier wanting to sell him products, and at the same time a competitor. Again a conflict: on one hand trying to serve the independent by selling him products; on the other hand, aggressively competing with him and taking business *away* from him.

We feel that, if we went into the captive laboratory operation, we would have too many compromises to make, that our contribution as a manufacturer would be reduced, that we would in effect simply be trying to sell direct.

Maybe you think you would prefer us to sell direct. Let's look at the advantages you have with the independent manufacturer-independent distributor system.

1—An independent laboratory distributor can offer you a broader product line and more comprehensive service. He can and does select products from many manufacturers, bring you the best that is available and those that are most suitable for you.

2—He screens products as they are introduced, and can counsel and detail you on the strengths and possible shortcomings of each product. He is not committed to support any one manufacturer. His sole interest is in serving you and helping you serve patients better.

3—One of the independent's functions is the stocking and inventory of product. A captive lab, to properly support its manufacturing arm, tries to sell you inventory, sometimes at special deals because its primary responsibility is to sell the products of the parent factory. A good independent supplier recognizes this inventory maintenance as his responsibility and serves you better because of it.

4—An independent distributor is *local*. He knows your problems because he's close to you, and, as a local business owner, he has the flexibility to meet your special needs. A captive lab is directed partially on a national basis and partially in a large regional system. Business practices are determined by executives who don't know you or your particular needs even if they do understand them.

5—We as a manufacturer can "keep sharp" by having to compete with other manufacturers in order to sell our product to the independent laboratory.

Unless your patients are pleased, you cannot exist in practice. Your independent distributor is your "silent partner," helping you to fulfill your responsibility. Only if that partner is devoted 100% to serving *your* needs can he be most effective. At Shuron, we will stand on our product advantages rather than a "captive" or direct selling system to market our goods. We want not to compete with your silent partner, because we would be injuring him. Our concern rather is to strengthen him, and so to strengthen you and the entire industry.

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OPTOMETRIC NEUROLOGICAL CASE HISTORIES*

Bertram L. Roberts†
Los Angeles, California

INTRODUCTION

The three cases briefly to be discussed today are taken from my office files to illustrate that patients with neurological disease can and do consult optometrists. I would like to state here to my optometric colleagues and to the ophthalmologists and the neurosurgeon present that I am quite humble about pathology and about the responsibility of clinical decisions. The cases I present, and the cases most of us present, are those in which we have helped uncover the nature of the problem. I should find it extremely discomforting to be confronted by those patients of whom perhaps I may be unaware, but in whom I have missed the evaluation completely.

The three patients we are going to consider may seem an unfortunate choice to present because all died not long after surgery. Most others in my past discussions had happier outcomes. That may be why these have remained unpresented. It should be stated that the first two patients probably had the odds in their favor surgically, but they didn't make it.

CASE #1

On April 27, 1949, a 47-year-old woman was referred to the office by her brother who was also our patient. The history is as follows: Her spectacles were broken. On her previous eye examination done elsewhere some six months before this visit, she had been told that she needed no change in these. However, her persistent headaches for the past six months were very discomforting and she wanted a consultation for this. She described her headache as being "back of my eyes" and at the "base of my neck." She had been having this headache almost daily and usually awoke with it. In response to questions about her general health, she said she was "anemic," had "low blood pressure," and was "nervous."

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*Read before the Los Angeles County Optometric Society, Spring Pathology Seminar, Los Angeles, California, April 12, 1961, following a neurosurgeon's discussion of "Ocular Manifestations of Neurological Disorders." For publication in the November, 1961 issue of the AMERICAN JOURNAL OF OPTOMETRY AND ARCHIVES OF AMERICAN ACADEMY OF OPTOMETRY.

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Her refractive condition was that of low hypermetropia and presbyopia. Corrected vision in each eye was 20/20. Gross confrontation was normal. The optic discs showed blurred margins, but no elevation, both eyes. The fundi were otherwise intact. My impression at the time was that the blurred disc margins were suspicious, but that this could well be a congenital anomaly.

The patient was requested to return the following day for a visual field examination. Examination with the tangent screen showed probable enlargement of the blind spots, the right measuring 18 x 20 cm. at one meter, the left somewhat smaller. The word probable was used because the fields were very difficult to map on this apprehensive patient. Cooperation for her on the field test was very trying. The patient was referred to an ophthalmologist for consultation.

The ophthalmologist reported that in his opinion the blurred disc margins were probably a congenital anomaly. He stated, however, that he, too, could not feel certain about it, and suggested that I see the patient on a few occasions in the future to note any possible changes. He felt that if the condition showed no change it could safely be considered a congenital anomaly.

On May 9, some twelve days after her first visit, the patient called for her new spectacles. A fundus examination at that time showed no apparent change in the appearance of the discs. Her headaches had not changed as regards frequency or intensity. It was at this point that I tried to get a more informative history from her. She stated that she had difficult equilibrium occasionally, that her memory was poorer, that she had some nausea and a desire to vomit at times although actually never did.

Although objectively nothing had changed, I found this history disturbing and discussed the case by telephone with a neurosurgeon. He was in agreement that the slightly blurred discs coupled with this history warranted a neurological examination.

The patient was a very nervous woman who had never been married, and the next day I discussed the problem with her nearest relative, her brother. When we discussed details of her suspicious history he indicated that he thought them not too significant, for she was always somewhat awkward in gait and absent-minded. I tried to persuade him to have her see the neurosurgeon nevertheless, explaining that her symptoms could have intensified without his being aware of changes.

He decided to wait as the ophthalmological consultant had not stressed any urgency in the matter, and he himself was not convinced of

the necessity. It was agreed that she would return in two to three months.

She appeared for examination three and a half months later, August 31, and the blurring of the disc margins had become more exaggerated. I then insisted upon a neurological examination.

On October 6, the neurosurgeon reported that he, in conjunction with an otolaryngologist was fairly convinced of the presence of an VIII nerve tumor.

I saw her on October 7, and the impression was of increased choking of the discs, but with scarcely any elevation. She had developed an occasional nystagmus in dextroversion.

The acoustic nerve tumor was removed in early November, but the patient developed edema of the medulla, a complication sometimes following such procedures and died on or about November 14 of post-surgical complications.

CASE #2

On May 14th, 1960, a male, age 59, presented himself, referred by a relative for routine eye examination with a view to changing spectacles. History is as follows: Occupation: Worked on a bench at assembly in a small plant. Chief complaint: "Can't read small print." In general health, he had no special complaints except for a tendency to be, in his words, "always sleepy." Attempting to read and viewing television were described as activities that were especially sleep producing. My impression at this point in taking the history was that the patient was a tired, hard-working presbyope who needed a refractive change urgently.

Uncorrected vision of the right eye was 7/400; of the left, 20/400. Retinoscopy of the right eye showed it to be close to emmetropia with no improvement in visual acuity with the low cyclinder found. Refraction of the left eye yielded 20/70 as the maximum acuity with $+2.00 \bigcirc -0.50 \times 90$, essentially the same prescription as he had been using. There was a slight rotary nystagmus, bilaterally. Fundi showed a marked optic atrophy of the right eye, and a pallor of the left disc. Gross confrontation showed a bi-temporal hemianopsia, which was more accurately demonstrated on the tangent screen. Figure 1. Impression: Probable pituitary tumor.

The patient had been examined elsewhere and had spectacles prescribed 2 years prior to this examination. Also he had passed the motor vehicle department drivers' test on August 11, 1958, some 21 months prior to showing up in this advanced state of visual deterioration. I think this would indicate a fairly rapid development of visual decline.

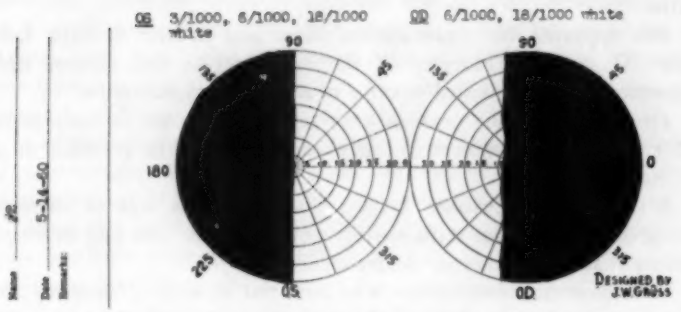

Fig. 1. Field chart showing bi-temporal hemianopsia.

The patient was urged to have a neurological consultation as soon as possible.

The probable diagnosis of chromophobe adenoma of the pituitary gland was made by the neurosurgeon. Surgery proved this to be correct. However, on June 2, 1960, I was informed that the patient had died following surgery. Now this was a fairly obvious case—actually it is amazing that the patient reached this condition and was unaware of any serious impairment until he had come in to have his lenses changed.

CASE #3

The next patient, a woman, age 65, was seen on June 9, 1958. She had been examined by an ophthalmologist less than two weeks before, on May 27th. The ophthalmologist had given her a prescription for new spectacles which she had not had filled and which proved to be essentially the same prescription she was then using. I did not mention this to the patient.

Her complaints were as follows: A feeling of a "cinder" in the left eye—also seeing flashes of colored lights with the left eye. She had been given medication by the ophthalmologist, but it didn't help. She had been referred to me by another patient and wanted further consultation in determining what was wrong. She had been seeing lights for six months. She reported her general health as being "alright," but that she was "extremely nervous." Additional visual history elicited the information that her eyes tired in watching television, and that print sometimes blurred in reading.

Refraction revealed that her own lenses were grossly adequate, but that she was spherically corrected and required an additional 0.50 D. cyl.

in the right eye and a 0.75 D. cyl. in the left. Her best visual acuity was 20/40, right eye, and 20/25 –2, left eye.

There were lenticular opacities in both eyes. The fundi were essentially normal with the possible exception of a slight pallor of the left optic disc.

In further discussion with the patient she recalled a recent episode of an error in judgment. She had been living in the same apartment for about 12 years. It had been her habit and the habit of other tenants of the building to go to the roof of the building for an exposure to fresh air and sunshine. One day she came down from the roof out of the bright sun and somehow unaccountably found herself trying to open the door of the wrong apartment with *her* key — the apartment was situated at some distance from her own. She was very upset by this experience.

Gross confrontation showed a dimness of the left inferior quadrant, both eyes, with hand movements. Central fields showed a left homonymous hemianopsia, very difficult to map with accuracy, but *definite*. Figure 2. The impression was a cerebro-vascular accident, or more likely an occipital or parietal lobe tumor.

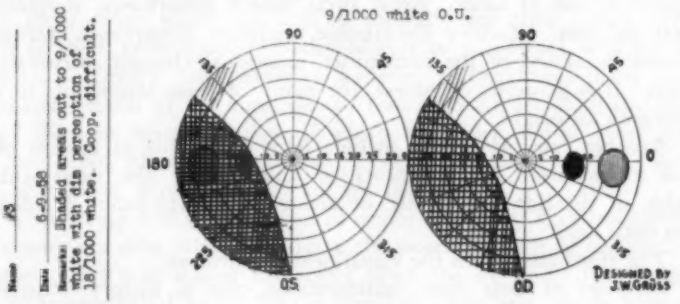

Fig. 2. Field chart showing left homonymous hemianopsia.

The patient was referred to a neurosurgeon. I never saw her again, and some of the rest of the story I learned later from the patient who had referred her. It seems that the woman's husband, whom I had never met, had been unhappy with my referral of her to a neurosurgeon and with my insistence that the possibilities could be serious, and he prevailed upon her instead to visit a different ophthalmologist one week later. This second ophthalmologist examined her, but apparently found nothing unusual, and suggested she return to her general physician for care. It is presumed that nothing neurological was sus-

pected by her general physician until he began noticing general neurological signs and hospitalized her for study. A neurological consultant was called in who suggested immediate surgery. She was operated on August 3rd or 4th.

Having been apprised by my informant of the identity of the patient's physician, I telephoned him and the neurosurgeon for a report. The diagnosis was glioblastoma multiforme of the parieto-occipital junction, right occipital lobe, a malignant tumor. On September 11, 1958, 3 months after my examination, she died.

CONCLUSION

Although neurology and the area that concerns us, neuro-ophthalmology, are complex studies, it behooves us to have some background in these fields.

Our chief responsibility in regard to neurology is that of screening patients with possible neurological disease.

There are, in general, three main categories of signs of the neurological suspect.

The first is history. We may have one or several of the following complaints: Headache, vomiting, convulsions, loss of memory, amenorrhea, loss of libido, visual auras, speech disturbance, sleepiness, mental dullness, olfactory disturbance, auditory disturbance, vertigo, dizziness, weakness of the extremities, emotional changes, personality changes. It is possible, of course, for many of these complaints to be psychogenic in origin.

Another category would include signs observable in routine objective examination. Impairments of function of the extra-ocular muscles, of the lids, the pupils, of accommodation; pallor or edema of the optic discs.

The third category is the visual field impairment.

Any one of these three considerations, that is, history, objective examination or visual field examination, can alone be significant in uncovering the neurological patient for referral. Any two or all three combined add up to definite evidence.

442½ North Fairfax Avenue
Los Angeles, California

OCULAR SIDE EFFECTS OF SYSTEMIC MEDICATION*

Philip G. Sloan†
Palo Alto, California

Nearly all chemical agents will cause pathological changes if applied to an eye in sufficient concentration for a long enough period. In addition, the accidental ingestion or inhalation of many compounds in toxic amounts has been shown to produce specific ocular damage. The literature of ophthalmic toxicology dealing with acids, caustic liquids, irritant gases, heavy metals, and certain hydrocarbons is vast and well known. Much less concern has been shown for the chronic and acute toxic effects of systemic medication. With the broad and rapid application of new and complex synthetic compounds this area is assuming increased importance.

This survey attempts to include the currently used systemic drugs which have been shown to have significant ocular side effects. The drugs are organized, where practical, by their therapeutic use. Within the groupings they are listed by generic name, with some examples of the more common trademarked preparations. For the purposes of this paper those drugs which are applied topically for their ocular effect (such as DFP, cyclopentolate hydrochloride or paredrine) will not be considered. Others (amphetamine, caffeine, and codeine) have been eliminated because of the minimal and transitory nature of their ocular effects. Finally, many compounds which were formerly of great concern (optochin, plasmocide, thallium, cystine, and dinitrophenol) have been abandoned as remedies and are not included.

Some drugs will show unwanted ocular side effects very frequently. For example, a drug which will relax the smooth muscle of the gastrointestinal tract will almost invariably affect the iris and ciliary body. The pharmacological action here is identical. Similarly, drugs used to poison intestinal parasites are often found to have some toxic effect on the host. Most drugs, however, show a low incidence of marked side effects. Often the signs and symptoms listed are found only in high dosages or in individuals unusually sensitive to the drug.

No list such as this can hope to be complete. Annually hundreds of new drugs are introduced and hundreds of new side effects are discovered. Each drug will only rarely cause unwanted actions or it will

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†Optometrist. Fellow, American Academy of Optometry.

soon be abandoned. However, as Tassman [1a] has pointed out, although the marked effects may be infrequent, there may be a much higher incidence of slight reactions giving rise to vague ocular symptoms. For this reason it becomes increasingly important to determine whether any medication is being taken by the patient. In addition to providing information of physical disorders which may relate to the eyes, it may also serve to uncover information that may help to explain unusual or bizarre ocular signs and symptoms.

I. ANALGESICS AND SEDATIVES

Aminopyrine (Pyramidon ®)	Optic atrophy, conjunctivitis.[28n]
Antipyrine (Felsol ®)	Urticaria of lids, tearing, transient amaurosis.[1b]
Barbiturates Amytal Barbital Dial Evipal Pentobarbital sodium Phenobarbital Seconal Sodium pentothal Veronal	Nystagmus, alteration of pupil size, hippus, extraocular palsies, transitory amblyopia, ptosis, blepharitis, diplopia, optic neuritis, chromatopsia.[2a, 28j]
Bromides	Visual hallucinations, macropsia, micropsia, chromatopsia, blurred vision, diplopia, altered pupil size, photophobia, reduced corneal reflex, conjunctivitis.[1c, 2b]
Carbromal (Adaline ®, Dormiturin ®)	Anisocoria, nystagmus, optic atrophy, scotomata.[25e]
Chloral hydrate	Miosis.[28h]
Cobra venom	Increased visual acuity, widened color fields.[3b]
Meperidine (Demerol ®)	Transient blurring, lid edema, mydriasis.[4a, 28k]
Methadone (Dolophine ®)	Miosis.[4b]
Methylene blue	Blue-green eye grounds.[2c]
Methylpentynol (Allotropal ®, Dormison ®, Somnesin ®)	Mydriasis, ptosis, nystagmus, diplopia.[24d]
Morphine	Miosis.[2d]
Paraldehyde	Visual hallucinations.[2e]
Phenylbutazone (Butazoladin ®)	Lowered visual acuity, pseudo-presbyopia, optic neuritis, chromatopsia.[24a, 27a]
Phenylethylhydantoin (Nirvanol ®)	Conjunctivitis, lid edema.[28x]
Quinine	See Antimalarials.
Salicylates	Amblyopia, mydriasis, lid edema, constricted visual fields, nystagmus, miosis, conjunctivitis, optic atrophy.[1d, 5a, 28g]

II. ANTHELMINICS

Aspidium (Felix mas)	Transient amaurosis, mydriasis, optic neuritis, xanthopsia.[1e, 6a]
Chenopodium	Amaurosis.[2k]
Pelletierine	Mydriasis, toxic amblyopia.[4n]
Piperazine (Antepar ®, Vermidol ®)	Partial cycloplegia, photopsia, chromatopsia.[24b, 27b]
Santonin	Xanthopsia, photopsia, retarded dark adaptation.[1d]

III. ANTIBACTERIALS AND ANTIBIOTICS

Arsenic—organic pentavalent (Tryparsamide, Carbarsone, Arsacetine)	Toxic amblyopia, keratitis, conjunctivitis.[7a, 28p]
Arsphenamines (Salvarsan, Neosalvarsan, Sulpharsenol, Mapharsen, Bismarsen)	Mydriasis, lid edema, corneal edema, panophthalmia, keratitis, conjunctivitis, corneal ulcers, myopia.[28q]
Chloramphenacol (Chloromycetin ®)	Optic neuritis.[3a]
Chlortetracycline (Aureomycin ®)	Optic neuritis.[28b]
Cycloserine (Seromycin ®)	Reduced visual acuity, paralysis of ocular muscles.[27d]
Furaltadone (Altafur ®)	VI N. palsy, diplopia, blurred vision, nystagmus.[9]
Isoniazid (Cotinazin ®, Isonicid ®)	Optic neuritis, myopia.[27e]
Neomycin	Blurred vision.[10]
Nitrofurantoin (Furadantin ®)	Itching, burning, tearing.[3e]
Paraminosalicylic acid (PAS)	Toxic amblyopia, photophobia, photopsia.[7b, 28a]
Penicillin	Blepharoconjunctivitis, ptosis, phlytenulae, subconjunctival hemorrhage.[28c]
Polymyxin (Aerosporin ®)	Diplopia, nystagmus.[28o]
Streptomycin and Dihydrostreptomycin	Xanthopsia, optic neuritis, partial cycloplegia, oculomotor disturbances.[5b, 8a, 27c]
Sulfonamides—especially: Sulfadiazine Sulfanilamide	Transient myopia, conjunctivitis, reduced accommodation and convergence, xanthopsia, retinal edema, optic neuritis, changes in muscle balance.[2f]

IV. ANTICONVULSANTS

Diphenylhydantoin sodium (Dilantin ®)	Conjunctivitis, diplopia, nystagmus, ptosis, transient amblyopia, visual hallucinations.[2g]
Ethotoin (Peganone ®)	Diplopia, nystagmus.[25b]

Paramethadione (Paradione ®)	Photophobia, hemeralopia, chromatopsia, toxic amblyopia, diplopia, conjunctivitis.[28l]
Phenacemide (Eferon ®, Phenurone ®)	Mydriasis, nystagmus.[25c]
Primidone (Mysoline ®)	Nystagmus, lid edema.[27g]
Trimethadione (Tridione ®)	Photophobia, hemeralopia, toxic amblyopia, chromatopsia, diplopia, conjunctivitis.[28l]

V. ANTIDEPRESSANTS

Etryptamine acetate (Monase ®)	Blurred vision.[26]
Imipramine (Tofranil ®)	Cycloplegia.[25e]
Iproniazid (Marsilid ®)	Cycloplegia.[24e]
Isocarborazid (Marplan ®)	Blurred vision.[20a]

VI. ANTIEMETICS

Chlorpromazine (Thorazine ®)	Blurred vision—see also Tranquilizers.[11a]
Cyclizine (Marezine ®)	Blurred vision.[11a]
Dimenhydrinate (Dramamine ®)	Blurred vision.[11a]
Diphenhydramine (Benadryl ®)	Blurred vision—see also Antihistaminics.[11a]
Meclizine (Bonamine ®)	Blurred vision.[11a]
Prochlorperazine (Compazine ®)	Blurred vision—see also Antihistaminics.[11a]

VII. ANTIHISTAMINICS

Generally	Blurred vision, diplopia.[4d]
Diphenylhydramine (Benadryl ®)	Partial cycloplegia, ptosis, hemianopsia, mydriasis, temporary amaurosis.[2h, 28e]
Prochlorperazine (Compazine ®)	Tonic spasm of ocular muscles.[24c]
Pyribenzamine	Partial cycloplegia, myopia, corneal edema.[12, 28e]

VIII. ANTIMALARIALS

Chloroquine (Aralen ®, Milbis ®, Triquin ®, Tritis ®)	Cycloplegia, keratopathy, "haloes," diplopia.[21, 28m]
Plasmoquine (Pamaquine)	Mydriasis, photophobia.[28l, 4m]
Quinacrine (Atabrine ®)	Scotomata, enlarged blind spot, corneal edema.[4e]
Quinine	Toxic amblyopia, nictalopia, diplopia, photophobia, mydriasis, chromatopsia.[4f]

IX. ANTISPASMODICS — SKELETAL MUSCLE

Benztropine methanesulfonate (Cogentin ®)	Blurred vision, mydriasis.[4o]
Caramiphen (Panparnit ®)	Partial cycloplegia, headache, diplopia, mydriasis.[4g, 3a]
Cycrimine hydrochloride (Pagitane ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29a]
Ethopropzine hydrochloride (Parsidol ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29b]
Mephenesin (Dioloxol ®)	Nystagmus, diplopia.[28f]
Methocarbamol (Robaxin ®)	Blurred vision.[25d]
Procyclidine (Kemadrin ®)	Blurred vision, mydriasis.[11b, 24d]
Tridihexethyl iodide (Pathion ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29c]
Trihexyphenidyl (Artane ®)	Blurred vision, mydriasis.[4h]

X. ANTISPASMODICS — SMOOTH MUSCLE

Generally	Mydriasis, cycloplegia, increased intraocular pressure.
Aminopentamide sulfate (Centrine ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29d]
Antipyrine	See Analgesics and Sedatives.
Belladonna Alkaloids	See separate category.
Diphemanil methylsulfate (Prantal ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29e]
Diphenylhydramine	See Antihistaminics.
Methantheline (Banthine ®)	Mydriasis, cycloplegia, increased intraocular pressure.[4j]
Oxyphenonium (Antrenyl ®)	Mydriasis, blurred vision, increased intraocular pressure.[4l]
Penthienate bromide (Monodral ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29g]
Propantheline bromide (Neodexabine ®, Probanthine ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29h]
Tricyclamol chloride (Elorine ®, Tricoloid ®)	Mydriasis, cycloplegia, increased intraocular pressure.[29i]

XI. HYPOTENSIVES

Bretylium tosylate (Darenthin ®)	Toxic amblyopia.[25a]
Chlorisondamine chloride (Ecolid ®)	Cycloplegia, mydriasis.[13]
Hexamethonium chloride (Methium ®, Esomid ®, Hexameton ®, Bistrium ®)	Cycloplegia, mydriasis, amaurosis.[14]

Mecamylamine (Inversine ®)	Cycloplegia, mydriasis.[13]
Nitrites (amyl nitrite, nitroglycerine)	Transitory "haloes."[4k]
Pempidine (Estomycin ®)	Blurred vision.[25i]
Pentaerythritol tetranitrate (Pentafin ®)	Increased intraocular pressure.[29f]
Pentolinium tartrate (Ansolysen ®)	Cycloplegia.[13]
Rauwolfia (Reserpine, Serpasil ®)	Miosis, ptosis.[18a]
Veratrum alkaloids (Veriloid ®)	Blurred vision.[4c]

XII. TRANQUILIZERS

Chlorpromazine (Thorazine ®)	Miosis, rare mydriasis, diplopia, cycloplegia, conjunctivitis, blepharitis.[22, 27f]
Meprobamate (Miltown ®)	Blurred vision, diplopia, mydriasis.[18a, 25j]
Perphenazine (Trilafon ®)	Blurred vision.[25f]
Rauwolfia	See Hypotensives.
Thioridiazine (Mellaril ®)	Blurred vision, cycloplegia, iridoplegia, pigmentary degeneration of the retina.[16]

XIII. VITAMINS

A	Mild exophthalmos, loss of lashes.[19]
B-1 (Thiamin chloride)	Conjunctivitis.[28t]
D-2 (Calciferol)	Optic atrophy.[28u]

XIV. BELLADONNA ALKALOIDS

Atropine	Cycloplegia, mydriasis, increased intraocular pressure.[6e]
Homatropine	Cycloplegia, mydriasis, increased intraocular pressure.[6e]
Hyocyamine	Cycloplegia, mydriasis, increased intraocular pressure.[6e]
Scopolomine	Cycloplegia, mydriasis, increased intraocular pressure.[6e]

This group represents a special category in two respects. First, these drugs are among the most widely used in medicine. They have long been used in ophthalmology; are used as respiratory stimulants; and in the treatment of cardiovascular, gastrointestinal, and urinary disorders, Parkinsonism, asthma, and coryza. The 1960 Edition of "Physician's Desk Reference to Pharmaceutical Specialties and Biologicals"[29] lists over 200 compounds in which one of these drugs is a principal ingredient. Second, since the primary action of these drugs is to relax smooth muscle, they exert a powerful effect on the iris and ciliary muscle. As a group, these drugs are probably responsible for more ocular side effects than any other.

XV. MISCELLANEOUS

Alcoholism	
Disulfiram (Antabuse ®)	Retrobulbar neuritis.[7a]

Arthritis	
ACTH	Transient myopia, lacrimation.[28s]
Gold sodium thiomalate (Myochrysine ®)	Ocular chrysiasis.[18b]
Asthma	
Ephedrine	Mydriasis, blurred vision.[3a]
Racephedrine hydrochloride	Mydriasis.[29j]
Stramonium	Toxic amblyopia.[3c]
Cardiac Disease	
Acetazolamide (Diamox ®)	Transient myopia.[25g]
Digitalis	Visual hallucinations, miosis, mydriasis, chromatopsia, transient scotomas, retrobulbar neuritis, flickering vision.[2i, 6b]
Quinidine	Blurred vision, photophobia, diplopia, scotomata, mydriasis, optic atrophy.[8b, 28d]
Diabetes	
Chlorpropamide (Diabenese ®)	Toxic amblyopia.[30]
Insulin	"Insulin presbyopia."[41]
Hyperthyroidism	(also in radiocontrast media and expectorants)
Iodides	Conjunctival irritation, keratitis, vitreous opacities.[23]
Intestinal paresis	
Physostigmine (Eserine)	Miosis, spasm of accommodation.[28v]
Menopausal disturbances	
Diethylstilbestrol	Partial cycloplegia.[17]
Mental Research	
Lysergic acid diethylamide (LSD)	Mydriasis.[15]
Migraine	
Ergotomine (Gynergen ®)	Toxic amblyopia, mydriasis, cataract, miosis, spasm of retinal vessels.[1e, 2j]
Nasal Congestion	
Naphazoline (Privine ®)	Miosis.[28r]
Tetrahydrozoline (Tyzine ®)	Miosis, mydriasis.[25h]
Peripheral Vascular Disease	
Dibenamine	Diplopia, miosis.[28w]
Tetraethylammonium bromide (T.E.A., Teamyl ®)	Cycloplegia, ptosis, mydriasis.[28w]
Sciatica	
Gelsemium	Mydriasis, ptosis, diplopia.[1f]
Urinary Retention	
Bethanechol (Urecholin ®)	Itching, burning, tearing.[3c]

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THE ROLE OF BINOCULAR STRESS IN THE POST-WHIPLASH SYNDROME*

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In its simplest terms, a whiplash injury is considered to be a product of a violet alternate flexion and extension of the head and neck following the impact of two automobiles. Usually this is the aftermath of the common rear-end type of collision and the extent of the injury is not directly proportional to the speed or force of the impact.

This whiplash syndrome is rapidly becoming a colossus in the medicolegal world. Such injuries of the spine are rapidly taking their place among the leading causes for litigation.

The very nature of a whiplash injury is conducive to pain and discomfort in the absence of visual evidence of bone, disc, ligament, or nerve trauma. When x-ray shows no change in these structures, the injury should respond to treatment within a reasonable period of time, or merely time itself would effect a cure; however, the evidence indicates that a great percentage of these patients complain of chronic pain for years after such an injury.

When there is no visible cause for pain, there are always those who immediately label such problems as psychosomatic. Many physicians and lawyers label the chronic post-whiplash syndrome as psychosomatic and maintain that the most effective cure is the "green poultice" applied by insurance companies.

It is the purpose of this paper to point out that many patients complaining of chronic severe headache, posterior cervical tension, and related symptoms following head or neck trauma are not malingerers or psychoneurotics but have a bona-fide problem which will respond to proper therapy.

HISTORY

In 1920, the Navy recognized that carrier pilots were sustaining cervical spine injuries as the result of being catapulted from a flight deck. The men would report sudden black-outs at the moment of violent acceleration and, if they were fortunate, would regain consciousness after becoming airborne. Many were not so fortunate. Symp-

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toms of cervical sprain developed and further injuries were prevented by the addition of a head rest to the cockpit seat.

As the automobile became more common, the accident rate began to multiply. More and more cases of cervical strain became evident. In 1934, Lownerblad[1] described a "hurling back syndrome" resulting in a snapping of the neck. In 1945, Davis[2] described the injury as we know it today. In 1949, Jackson[3] reported a "cervical syndrome," but it was not until 1950 that it appeared in literature to any extent. In 1953, it was popularized by Gay and Abbott.[4] Little is noted in foreign journals, as whiplash appears to be peculiarly American, with its primary incident related to the heavy, high-speed automobile, driven in congested areas.

The incident of whiplash injuries to the neck from automobile accidents has increased at a tremendous rate. In 1959, there were approximately 1,400,000 disabling injuries[5] in motor vehicle accidents and, of these, 15 per cent were of the whiplash type. This means that in 1959 approximately 210,000 people sustained anywhere from mild to severe cervical injury from motor vehicle accidents alone.

DESCRIPTION

In demonstrating the force applied to the neck in such an accident, McLaughlin[6] has shown that when a 3500 lb. car traveling at 10 m.p.h. strikes the rear of another car, it can transmit a force of twenty-five tons. The person's body in the car being struck continues to move forward, while the head, being hinged at the neck, snaps backward. The average head weighs eight to twelve pounds, and the cervical vertebrae are very delicate. The force that is pushing the head backward is even greater than believed, since the neck acts as a fulcrum and the leverage is applied near the top of the head. As a result, the head snaps back, with the equivalent of several tons of force, without any support, since the muscular control of the neck is caught off guard. There may even be more than one oscillation of the head and neck, especially in a three-car collision.

Small boys have found a sharp snap of the neck a very convenient method to decapitate small snakes. Mark Twain's Tom Sawyer and Huckleberry Finn used this method and referred to it as the whipsnap.

SYMPTOMATOLOGY

On the basis of the above analysis of the resultant force which produces the injury, it can readily be seen that extensive damage may result. Weinberg[7] believes that a concomitant concussion of the brain may be produced by the acceleration-deceleration mechanism. Nielsen,[8] Hackett,[9] and others accentuate the spinal ligament and tendon sprain.

Billig[10] has made an analysis of observable injuries and listed many such as narrowing of the intervertebral discs, chip fractures, compression fractures of the vertebrae, ligament tearing, and involuntary muscle spasm.

The results of such injuries are found in symptoms of headache; posterior cervical tension; trapezius tension; vertigo; blurred vision; hyperesthesia; referred pain to arms, fingers, forehead, nose, eye, temple, and parietal region of the head; nausea; motor and sensory loss. Nielsen[8] reports two cases where there was a complete loss of knowledge (memory) of life experiences without loss of intellectually-learned facts. This type of affection has been shown to result from lesions of the hippocampal gyri, which lends proof to the probability of intracranial trauma.

OCULAR CHANGES

That head trauma can produce heterotropias is by no means a recent observation. This has been well known and observed down through recorded medical history. That head trauma can produce heterophorias is, of course, a later observation since phoria testing has been available; however, that injuries to the cervical spine can induce latent binocular stress is a most recent observation.

Allbutt, in 1870, first called attention to the changes in the fundus in injuries to the spinal cord. Posey and Spiller[11] in 1906 showed that the cervical sympathetic was frequently involved in traumatic lesions of the spinal cord. They describe the ocular phenomena as consisting of: (1) changes in the optic nerve; (2) sympathetic phenomena; and (3) pupillary changes.

Billig,[10] analyzing a series of cases of whiplash, referred to a symptom of "blurred vision unrelated to ocular visual defects." He said, "This was complained of in nearly all of the cases. In those of severe involvement the blurred vision was persistent, and those in which the involvement was milder it was described as transitory." He recorded one patient's comment, "I seem to lose my focus."

Frankel,[12] in observing eye changes following cervical trauma, has this to say: "*Eye* — pupils of the eyes and the associated structures can provide many valuable clues in the diagnosis of residual trauma from injuries about the neck. Interruption or dysfunction of the sympathetic pathways to the head may produce Horner's Syndrome: (1) dropping of upper eyelid, (2) constriction of pupil, and (3) loss of ability to tear or water. Patients with so-called whiplash injuries frequently complain of blurred vision or difficulty with focusing. Dilated pupils and a flattened lens may result from sympathetic stimulation. The study of pupillary oscillations with the slit-lamp may well become a routine

office procedure when the realization of the part that eye changes play in evaluating neck injuries becomes more apparent."

Sales Vasquez[13] and associates reported three cases in which active spondylarthrosis produced, in addition to a cervicobrachial syndrome, visual disturbances, with changes in the field of vision and fundi.

HEAD, NECK, AND EYE REFLEX

The neck is a major organ of total body posture and orientation in space, along with the vestibular apparatus and the extraocular muscles. Schlesinger states that any type of neck disorder, whether static or dynamic, is capable of secondarily altering the postural information being fed centrally from the neck. He further states that vertigo and nausea are not necessarily evidence of a psychosomatic component in neck disorders but may well represent a specific organic response to abnormalities of neck dynamics.

That the organism's desire to maintain single binocular vision is also a major force in neck dynamics has long been shown in ocular palsies. In the face of a paresis of a vertically acting extraocular muscle, there must be a compensatory torticollis to alleviate the resultant diplopia. Here is a condition of the neck being forced into an abnormal position with its resultant gravitational pull, to be able to compensate for a binocular anomaly. Thus we see that lesions which create binocular stress will, in turn, cause resultant stress in the neck.

The importance of the neck in the role of a sort of gyroscope in spatial orientation is not always appreciated. The tonic neck reflexes, along with otolith organs, play a major role in the machinery of postural set. Duke-Elder[4] emphasizes this head, neck, and eye reflex:

> ". . . there is an intimate association between movements of the eyes and changes in posture, and a close functional reflex correlation exists between the extraocular muscles and the labyrinths which record movements of the head in space, and the muscles of the neck which register movements of the head with respect to the trunk.
>
> "In the normal condition all these reflexes are summated, the one supplementing the other, with the result that there is an extremely well-developed correlation of ocular, labyrinthine, and neck reflexes, by means of which, both in movement and at rest, and in the various physiologically possible positions of the head with respect to the body in space, the correct visual attitude and the suitable correlation of the two eyes are ensured.
>
> "Whatever be the exact mechanism, it is known that each labyrinth is associated with all the muscles of each eye."

Eckardt, McLean, and Goodell[15] have shown with electromyographic evidence that a continued abnormal stress on the oculorotary muscles will result in neck muscle spasm which can be the basis of severe neck pain.

In the experiments, a three diopter vertical prism was placed in front of the subject's eye in whatever position caused the most discom-

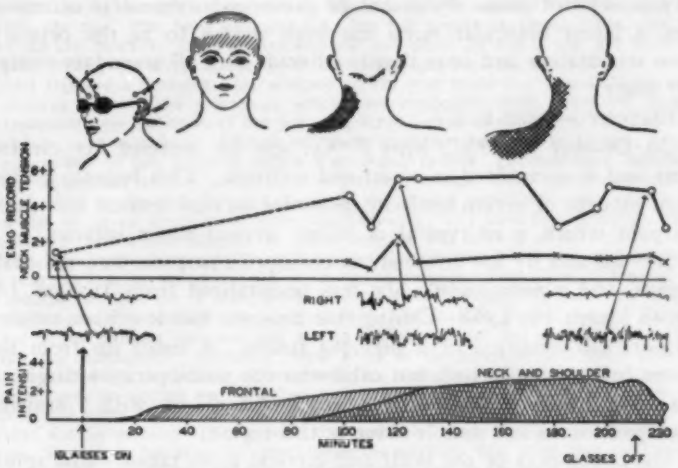

Fig. 1. The effect of abnormally sustained contraction of the external ocular muscles on the neck and head muscles. From *Headache and Other Head Pain* by Harold G. Wolff, by permission of the Oxford University Press, 417 Fifth Avenue, New York 16, N. Y.

fort. Diplopia was produced for about three minutes in all experiments. Electromyographic leads went to the frontalis, cervical, and trapezius muscles to record secondary muscle tension in these areas. The results show tension in the frontalis occurring within ten minutes; in the cervical area, within eighty minutes; and down the trapezius into the deltoid, within one hundred twenty minutes. The writers even describe the "stiff" muscle as definitely firmer to the touch and more bulky than its homologue.

Upon removing the vertical prism, this muscle spasm and discomfort lasted in some subjects up to several hours. It was shown in these experiments that neck massage gave temporary alleviation to the tonic spasm but soon afterward the tension increased to the original frequency.

This of course emphasizes that symptomatic relief of any referred pain can only be a temporary relief. It would be impossible to permanently eliminate a secondary pain unless the primary noxious stimulation is found and eliminated.

LATENT BINOCULAR STRESS

In the event of many of these post-whiplash syndromes, it has now been found that the physiotherapy, traction, and pharmacotherapy designed to relax the posterior cervical tension are only treating a sec-

ondary or referred pain. By means of prolonged monocular occlusion studies, a latent binocular stress has been shown to be the primary noxious stimulation and once that is relieved, then all secondary symptoms[16] abate.

CASE HISTORY — MISS F. G.

On October 4, 1957, Miss F. G., age 26, stopped her car for another and was involved in a rear-end collision. This began a period of eight months of severe headache, posterior cervical tension and associated pain which is so typical of many cervical strain injuries.

She was seen by her internist, an orthopedic surgeon, two ophthalmologists, and a neurologist. She was hospitalized from January 14, 1958, to March 19, 1958. During this time she had low back surgery to explore the condition of a previous fusion. A small lip from the previous fusion was excised, but otherwise the post-operative diagnosis was a severe sprain of the joint between L-3 and L-4, with a bruising of the ligamentous and muscle tissue in this region.

She had x-rays of the skull and cervical spine taken. The spinal x-rays were lateral, oblique, and while in brace. The radiologist's report was, "No pathological process seen and no localized structural changes. The cervical curve now appears about normal. The disc spaces do not seem unusual. The Cranial Vault is normal in appearance."

She received very extensive pharmacotherapy, including a number of analgesics, vasoconstrictors, and tranquilizers. She was intermittently put in traction to relieve the cervical spasm and good use was also made of physiotherapy. This was in the nature of microtherm, massage, fomentations, neck halter, and, finally, a neck brace upon dismissal.

Perhaps one of the most complete consultants' reports was that of the neurologist. It is herewith reproduced in toto:

> The following is my report on Miss F. G., who was seen for the first time at City Hospital on February 17, 1958:
>
> CHIEF COMPLAINT: Headache.
>
> FAMILY HISTORY: The patient's father is living but has arthritis. Mother and two sisters are living and well. She has never married. There is no allergy or migraine in the family.
>
> PAST HISTORY: The patient has had measles, mumps, chickenpox in childhood; she had rheumatic fever at the age of nine. She suffered severe headaches from 1953 until 1955. These were believed to be due to overwork and tension. She was found, however, to be allergic to about forty foods and ten other things. She is still taking shots for this and her headaches were improved. She had a tonsillectomy, appendectomy, and had a benign tumor removed from the breast. In 1950 she had a spinal fusion. She has had no serious injuries, does not smoke or drink. Her menstrual periods are normal except for slight irregularity for which she is taking cytomel.
>
> PRESENT ILLNESS: The patient states that aside from the headaches for which she has been treated with allergy shots, she was well until October 4, 1957,

when after going through an intersection, she stopped for another car, when she was hit from the rear. Her head was snapped. She felt a pain in her tailbone. She got out of the car, however, and took information and drove on and it was not until she had been driving for about fifteen miles that she began to have pain all over in her arms and legs and a headache. She stopped on her way home at a friend's home who is a masseur and received a massage which gave temporary relief. She had another such treatment over the weekend but her neck ached and it hurt to turn her head. She returned to work as a dental assistant but about this time developed the Asiatic flu and had a good deal of low backache which is attributed to this. Her headaches continued. She had an elevated temperature off and on for three weeks. When she started back to work, she had severe headaches and backache and x-rays were then taken and it was found that the spinal fusion done some years previously may have been damaged. After physiotherapy and a back support to relieve her, she was advised to have the back re-operated, which was done. However, she states that she has continued to have headaches near the base of the skull and in the frontal region. Her vision is blurred. It feels blocked out, but she is never sure. She states that immediately preceding coming into the hospital, her headaches were very severe. She could not sleep and she blacked-out for fifteen minutes and was sick for an entire evening, but she did not vomit. She has had momentary "blackouts" since being in the hospital. She states that she gets a dizzy feeling in the top of her head. She staggers to the right. Her headache has been worse on getting up. She had neck traction applied eight or nine days ago which helped some but did not completely relieve her headache. On questioning she says that she has vomited at times with the headaches. At times, she has a ringing in the ears. Sometimes her arms feel numb since the accident. She states that she has had trouble hanging onto things and has dropped instruments in her employer's office. She also complains of twitching in the left arm on occasion but not all the time. She states her back is much better since surgery. At times her vision is blurred and she has trouble focusing and this has happened three or four times after surgery. She has had trouble hearing and understanding what people say but no real loss of hearing.

GENERAL EXAMINATION: The patient is intelligent, cooperative, does not appear ill. Tonsil tags are present; teeth are well cared for. Blood pressure is 110/70. She is wearing an abdominal support and her abdomen is not examined. Breast and chest examination are negative.

NEUROLOGICAL EXAMINATION: A complete neurological examination was done and the following were noted: The biceps, triceps and supinator reflexes were bilaterally decreased −2. The patient was able to hear the watch tick on contact with the right ear (20 cm. normal).

ELECTROENCEPHALOGRAPHIC REPORT: A sixteen electrode electroencephalogram was taken on this patient February 13, 1958 at 1:00 p. m., and the following is my impression:

"IMPRESSION: Normal electroencephalogram. The patient exhibited a rather marked tendency to drowsiness."

OPINION: I believe this patient's headaches are on the basis of a tension state. I do not believe she suffers from any organic intra-cranial disorder. I think with constant reassurance she will make a good recovery. I believe there is a great deal of functional overlay in this case.

Because of the eventual outcome of this case, one ophthalmologist's report is herewith reproduced. The second ophthalmologist, in essence, gave a similar report.

HISTORY:

1. Constant headaches.
2. Blurred vision periodically.
3. Difficulty in changing focus from distance to near and vice versa at times.
4. Pain—sharp, shooting to dull ache accompanied by nausea at times through upper inner aspect of orbits—outside of eyes.
5. Double vision occasionally.
6. Patient also has had difficulty keeping her mind on what she was doing on occasion since the accident.

EXAMINATION:

E. O. M. (Extra Ocular Muscles) Normal in all excursions.
Fundi—Normal in both eyes.
Vision—RE 20/20+ LE 20/20+.
Phorias—distance—normal
near–6Δ–4ΔExo.
Visual Fields—Found to be normal in both eyes.

IMPRESSION:

1. Occasional double vision may be due to her exophoria.
2. The other complaints listed above (1, 2, 3, 4, 5 & 6), in fact, all probably could be attributable to the after effects of the accident—however, there are no eye findings pathological or otherwise to account for her complaint. She is a myope and her present correction is adequate.

As can be seen from the above reports, this patient did not reveal any pathology or functional problem as related to the accident, per se. For this reason, the consultants suggested the post-whiplash syndrome was from an anxiety tension state, with hysterical overlays, and that psychiatric help be secured.

Her physician, however, by the latter part of February had begun a trial series of monocular occlusion. His entry in the patient's hospital record on March 5, 1958, showed that the first noticeable relief was obtained by bandaging the left eye for one day. The relief was very slight but was at least the first clue of a binocular stress. There was no relief when the right eye was occluded nor when both eyes were occluded. She was, therefore, referred for an investigation of a possibl latent binocular stress.

Miss F. G. was first seen by the author on May 6, 1958. She was wearing a neck brace which had been a constant companion since she had left the hospital seven weeks previously. The pain in the cervical region was so intense when the brace was removed, that all of the findings on the first three visits were taken with the brace in place. Her current lens Rx which was one month old was:

O. D. $-1.25 \bigcirc\!\!\!

The new subjective was made up in temporary lenses to use while going through a standardized prolonged monocular occlusion routine.[17] On May 10 the patient occluded her left eye and kept it bandaged until the morning of May 13. She reported much less headache these three days, so that she was able to cut her analgesic intake to about one-half. The cervical tension was better and she could even leave the brace off for short periods. Her findings now showed:

Distance Phoria: 4Δ Exophoria
Vertical Phoria: 2Δ Left Hyperphoria
Vertical Ductions: O. D. Supra −1Δ/−1½ Δ Infra 3Δ/2½ Δ
O. S. Supra 3Δ/ 2½ Δ Infra −1Δ/−1½ Δ

Immediately following this test the right eye was occluded and remained under occlusion for three days. On May 16 she reported the headaches were still keeping down somewhat, but her vision was blurred at times and it created more general tension. She was still able to remove the neck brace for short periods. Her findings now were:

Distance Phoria: 8Δ Exophoria
Vertical Phoria: 1Δ Right Hyperphoria
Vertical Ductions: O. D. Supra 2Δ/1½ Δ Infra 0Δ/−½ Δ
O. S. Supra 0Δ/−½ Δ Infra 2Δ/1½ Δ

With the vertical phoria showing 1ΔR. H. following occlusion of the O. D. and 2ΔL. H. following occlusion of the O. S., it was decided to begin the temporary prism clip-over with 1Δbase-down on the O. S. and 2Δbase-in. This she wore for one week.

On May 23 new findings were taken after she had worn the temporary prisms constantly. She was instructed to close her left eye whenever she removed her glasses or clip-overs, so that at no time would a compensatory stress be set up in the binocular act. She now reported a considerable improvement in the headache intensity; in fact, so much so, that, by taking a few analgesics, she was free of headache until very late in the day. She was so much relieved of the posterior cervical tension that she was removing the neck brace for several hours at a time. She was now able to ride in a car without an increase in headaches and without the nausea she had previously had. There was still a mild vertigo, but this was only at short periods. Her findings now showed:

Distance Phoria: 5Δ Exophoria
Vertical Phoria: 1½ Δ Left Hyperphoria
Vertical Ductions: O. D. Supra −½ Δ/−1Δ Infra 2½ Δ/2Δ
O. S. Supra 2½ Δ/2Δ Infra −½ Δ/−1Δ

Her temporary prism clip-overs were changed to 1½ Δbase-down over O. S. and 3Δbase-in, and she wore these until May 28.

She reported some difficulty in adjusting to the new prism the first day, but by the second day it cleared up and the remainder of this trial period was even more asymptomatic than before. She was now

going without the neck brace for long periods and, in fact, on the 27th did not wear it all day. Her findings on this analysis were a duplication of those taken on May 23, so the following lenses was made:

O. D.: $-1.50\bigcirc-1.25 \times 150\bigcirc 1\frac{1}{2}\Delta$ In
O. S.: $-1.00\bigcirc-1.50 \times 20\bigcirc 1\frac{1}{2}\Delta$ In & $1\frac{1}{2}\Delta$ Down

After this lens prescription was delivered to her, she removed the neck brace permanently and has had no need of it since. By June 23 she began reporting an increase in the headache frequency and intensity. She was instructed to occlude the left eye for one day so new findings could be made. This was done on June 25. She now showed another $\frac{1}{2}\Delta$ increase in the left hyperphoria and another $1\frac{1}{2}\Delta$ increase in the exophoria, so new clip-overs of $\frac{1}{2}\Delta$down O. S. and $1\frac{1}{2}\Delta$In were put over her lenses.

By July 3 she reported complete cessation of headaches. She was taking no analgesics, and there was no feeling of posterior cervical tension. She was busy and active and leading a normal life. Her final lenses in a No. 1 tint were:

O. D.: $-1.50\bigcirc-1.25 \times 150\bigcirc 2\Delta$ In and 1Δ Up
O. S.: $-1.00\bigcirc-1.50 \times 20\bigcirc 2\frac{1}{2}\Delta$ In and 1Δ Down

SUMMARY

It is the purpose of this article to point out that:

1. A great percentage of the patients complaining of severe headache, cervical tension, and related symptoms following cervical trauma are not malingerers or psychoneurotics but have a bona-fide problem which will respond to proper therapy.

2. A severe cervical strain can produce a heterophoria, which can be found by using a standardized prolonged monocular occlusion test.

3. After the binocular stress is alleviated, the secondary syndrome will gradually dissipate.

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ASTHENOPIA AND ORTHOPTICS*

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The ocular disturbance, known to the layman as eyestrain occurs under many guises, accompanied by a variety of complaints. To the optometrist this condition is known as asthenopia. It is a distinct anomaly known to the profession for the better part of a century. Knowledge about asthenopia came as a result of discoveries in the field of ocular physiology, neurology, anatomy and physiological optics.

In 1843, Mackenzie introduced the term asthenopia in a paper entitled "On Asthenopia or Weak-Sightedness."[1] Curiously, he was not fully aware of the actual defect about which he was writing. Also, asthenopia was known under different names to the ancients. It was the Greek physician, Demosthenes, who wrote:[2]

> Eyes are said to be atonic when they can't bear to look at whiteness or redness (fiery things) but on the least excuse close the lids and water, especially when reading. And these differ from running eyes because running eyes shed tears without any external excitation, but these (the atonic) do not run without some exciting cause. The treatment is ambulatory, walking, running, gymnastic exercises, etc.

Mackenzie defined asthenopia as:[1]

> That state of vision in which the eyes are unable to sustain continued exercise upon near objects, although the patient, on first viewing objects generally sees them distinctly, although he can employ his sight for any length of time in viewing distant objects, and although the eyes appear sound.

Mackenzie had good reason to be concerned about asthenopia and the debilitating discomforts the condition produced. The late Walter B. Lancaster, himself very much interested in the subject, made a thorough study of asthenopia, both from the ophthalmological standpoint and the effect it had on shaping the lives of those afflicted. He tells of the fate of those who suffered from this debility in the year 1842, when little was known of the causes.[3]

> In 1842, if one's great-grandfather, then living in England, has suffered from asthenopia, what would have been his fate? He would have consulted one of the leading ophthalmic surgeons, perhaps Mackenzie or Lawrence or Adams. The physician, after examining and questioning the patient, would have noted certain facts which he regarded as important. The patient was, say, 18 years old and a student at Oxford, with good general health and a good family history. The eyes showed no signs of inflammation, present or past, and slight photophobia. There was a tendency to frown. Vision was very good for distant objects and was good for near for a short period. In reading, the print soon became blurred. A few months before, shortly after under-

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taking some special work involving study of old manuscripts much of the time by candlelight, the patient noticed the blurring of vision. At first it appeared toward the end of the week, after several days of steady use of the eyes. Resting the eyes over the week-end restored the sight. By degrees, the blurring came on earlier in the week. A few weeks before, the patient could read for over an hour, but lately the sight failed in a few minutes, so that he found it impossible to study.

Probably no record would be made of ocular discomfort, headache or other symptoms, local or general, except loss of sight, because at that time such symptoms were not regarded as a part of the picture of asthenopia.

The important questions which the physician tried to answer were: What brought on this serious condition and what should be done about it? He found that the occupation, study of old manuscripts, was the immediate cause. But many students were able to do this work: there must be some underlying cause and some contributing factors. There had been no acute illness to cause debility; no previous attacks of ophthalmia, especially no scrofulous inflammations of the eyes, and no cerebral symptoms (convulsions). The physician therefore fell back on the explanation commonly given in such cases, that the debility behind the ocular weakness was due to masturbation and spermatorrhea.

The prognosis given was the probable development of amblyopia and even amaurosis.

The treatment advised was: (1) cauterization of the urethra (to which Mackenzie devoted considerable space in his textbook)[4], (2) giving up of all use of the eyes for near work and (3) operation, which was mentioned as advocated by some surgeons but was not urged. Spectacles were not advised.

Some days later, while the patient was telling a friend about his sad affliction, his friend interrupted him: "I know just such a case. A young woman, a semptress who works for my aunt, Lady T——, became so blind that she could not see to thread her needle or do her sewing. She went to Lenz, the optician, who sold her some glasses, and now she is working as well as ever. You must see the optician at once. I have no doubt he can cure you."

So the patient went to the optician and was sold a pair of convex lenses, with which he found to his amazement that he could read all he wanted without the horrible blurring coming on. He resumed his work at the university with a profound feeling of relief.

A few months later, not having his glasses with him, he started to do some reading and found that he could not see a word! In a panic he went at once to the physician. The latter told him that what had happened was just what was to be expected. Wearing convex lenses inevitably led to just that loss of sight which he had suffered. It was a sure road to amblyopia. He should give up the glasses and take a prolonged rest in the country, or better still, emigrate to Australia or Canada and take up sheep raising or lumbering. His sight might then be restored . . .

Understanding the defect known as asthenopia, its causes and complications, has made considerable strides since the 1840's, and the days of Mackenzie, Lawrence and Adams. It is known that visual acuity is rarely directly affected and that amblyopia is not a resultant complication. And even if vision is impaired, as it might be from other causes, it might be restored to its former state, or even better, by proper therapy. Today, the causes of asthenopia are common knowledge. The functional impairment of vision which asthenopia may produce, lends itself to complete amelioration quite readily. The treatment may be either optical or orthoptic, or a combination of both; the use of convex lenses is no longer frowned upon. But then, it must be remembered that Mackenzie, Lawrence and Adams flourished and practiced their brand of ophthalmology in the first decades of the 1800's. Scientific in-

terpretation of visual and neuro-muscular physiology, physiological optics and ocular physiology and even some anatomical concepts, and, especially the function of the accommodation and its relationship to other visual functions, had to wait for Helmholtz, Donders, von Graefe, Hering, Tscherning, Worth and Maddox, Javal, Risley and Thorington, and other great minds of the middle 19th and early 20th centuries.

One can hardly blame Mackenzie for his thinking. He followed the prevailing and accepted concepts of his day from which few dared to deviate. He followed the thinking of his predecessors and was cautious not to incur the censure of his contemporaries whose thinking, like his own, followed an established mold. As Nicholson[5] so pithily stated: "When a tradition once becomes established by the hall-mark of acknowledged authority, it takes more than cold facts to uproot it from men's minds—it takes time."

Mackenzie, like his contemporaries and those who preceded them, regarded asthenopia an *ocular disease*. And since the eye tissues showed no signs of pathology and since no visible systemic or other disease was manifested, the nature of asthenopia and its cause or causes, was left entirely to conjecture and to the imagination.

Operation being one of the therapeutic means used in "correcting" or relieving asthenopia in the early 1800's, it is interesting to note the type of operation performed and the reasons given for its performance. The theory prevalent in those days was that the extraocular muscles were responsible for the action of the accommodation. Therefore, it was reasoned, that cure could be obtained by tenotomizing the recti muscles. Reports of cures from this method were frequent. Whether this was due to the enforced rest of the eyes and the accommodation, or whether alteration in effort by the convergence was the reason, is a matter of conjecture. But cures were reported.

It is important to note that only a few years elapsed between Mackenzie and Donders. To Donders[6] belongs the distinction of giving asthenopia its proper definition and pin-pointing its causes. In his great work on refraction and accommodation, Donders stated: "It is a great satisfaction to be able to say that asthenopia need now no longer be an inconvenience to anyone. The discovery of the simple fact that asthenopia is dependent on the hypermetropic structure of the eye, pointed out the way it is to be obviated." Donders revolutionized the treatment of anomalies of refraction and accommodation with which even the most skeptical could find no fault.

Having been shown the way, those who followed Donders lost no time in adding to the accumulated knowledge and supplementing it with clinical confirmation.

PRESENT KNOWLEDGE OF ASTHENOPIA:

From the clinical standpoint, there are several types of asthenopia. Asthenopia is not a specific ocular anomaly, but rather an accumulation of subjective symptoms with distress, headache, ocular pain or fatigue, predominating. Sometimes, visual acuity at distance and near may also be affected if the ciliary muscle is in a state of spasm. The refractive error, if one is present, plays an important role both in the symptomatology and in the manner of visual perception.

We now know that sometimes more than one type of asthenopia may occur at the same time in the same individual. Accommodative asthenopia may be complicated with a convergence defect, making it an accommodative-convergence asthenopia. Also, headaches may occur, adding cephalalgic asthenopia. If the patient has anisometropia, another element enters into the picture. This can be magnified in many ways by the addition of other non-related symptoms and conditions. It is most important that proper diagnosis is made and the defect correctly determined. Otherwise, only a part of the disturbance will be corrected.

Accommodative asthenopia, described so well by Donders, is by far the most common type. It may occur in combination with a disturbance of convergence, or it may be due entirely to accommodative impairment. Also, it may have some symptoms belonging to other types of asthenopia. However, the important clinical factor is that the accommodation is impaired, affecting functional performance of the visual apparatus.

The Dictionary of Visual Science[7] lists the following types of asthenopia: Accommodative, Accommodative-Convergence, Aniseikonic, Anisometropic, Aphakic, Astigmatic, Cephalagica, Cyclophoric, Dolens, Heterophoric, Hyperphoric, Hysterical, Integrative, Irritans, Motor, Muscular, Nervous, Neurasthenic, Presbyopic, Reflex, Retinal, Tarsal. Other clinical variations may be added to these. For example, the emotions may cause asthenopia, which may manifest symptoms similar to other types, yet belonging to no specific one.

With the advent and popularity of contact lenses, an additional type of asthenopia may be added to the already over-flowing list. Contact Lens Asthenopia, like the emotional type, has symptoms similar to several other types. Burning, ephiphora, headaches, ciliary pain, photophobia and photopsia and variable vision predominate. Yet, no objective or subjective signs or symptoms are observable in many instances. The contact lenses may have good physical fit and centering; slit lamp and fluorescein tests may be negative. One basis for the re-

action may be that while the patient has an urge to wear the lenses, he or she is extremely apprehensive of having a foreign body in the eyes.

Emotional asthenopia, also, has a hodge-podge of symptoms, the pattern of which cannot be made to fit into one specific or classical variety, as is the case, for instance, with the accommodative or convergence types. Here the slightest symptoms are exaggerated and magnified. The headaches are described as "splitting": mildest reduction in visual acuity is described as "blind as a bat," even though vision tests show the acuity to be 20/20, or a shade under it. This type may be synonymous with hysterical asthenopia, yet there is, infrequently, a clinical variance between the two. Happily, both types are known to respond favorably to visual training. Such a procedure may be a placebo. However, in some instances, the placebo, whether a sugar-coated pill, a pink sugary liquid, or visual training exercises, has a distinct place in the healing arts.

One type of emotional asthenopia may be classed as idiopathic. It is a self-originated condition, neither sympathetic nor traumatic. This type, too, may have hysteria as a basis with even deeper underlying etiological factors. Sometimes nutritional defects cause asthenopia. The symptoms may vary with the causal factors, with cephalagic predominating. Asthenopia Dolens (Dolorosa) is often associated with the instillation of miotic drugs in the eye.

The examination of patients with the symptoms of asthenopia consists of two parts—refractive and orthoptic. The refractive examination is primarily to obtain the case history and to determine the refractive state of the eyes. If the patient has simple hypermetropia or astigmatism, or compound hypermetropic astigmatism, optical correction is definitely indicated, especially for near use. There seems to be a tendency to recurrence of symptoms in persons prone to ciliary spasm. It is therefore advisable, as soon as the condition is discovered, to try to eliminate the cause. This is a preventive measure. Optical correction alone may not suffice. This can readily be determined during the second phase of the examination, the orthoptic. In myopia with ciliary spasm, minus optical correction is best deferred, or withheld altogether.

The orthoptic part of the examination must consist of the following steps:

The cover test, convergence near point, break and diplopia points, motility and comitancy tests, accommodation amplitude and reserves, muscle balance tests at distance and near, duction tests, fusion and fusional reserves, visual field tests, and, where indicated, tests for retinal

correspondence and spatial sense. The tests outlined are basic and required. Others may be necessary to establish a more definite diagnosis. Added to this array of tests may be an examination for aniseikonia, field studies or other special tests.

The designations given to the various types of asthenopia, invariably also define the basic cause for the respective condition. The treatment of asthenopia may be divided into two parts: the basic and the specific.

The basic treatment consists of methods directed toward relieving the symptoms. To be more comprehensive, let me cite the example of a patient with simple ciliary spasm.

The symptoms are: variably reduced visual acuity at distance. At times vision is normal: at other times objects at distance are blurred. Near vision is difficult. Headaches occur when the eyes are used. The complaints are present only when the eyes are used for any length of time, especially at close range. The patient usually has no spectacles.

Refractive examination shows emmetropia. The ciliary muscle shows spasm. The amplitude of accommodation is below average for the age. Convergence is ample, but there is strain in binocular fixation and the break point varies as does the diplopia point. Muscle balance tests show orthophoria at distance; low esophoria at near. Fusion is present, but the function is erratic and unsteady. The history substantiates the suspicion of ciliary spasm. The tests confirm this.

The treatment consists of two major steps: (1) Relaxation of accommodation; (2) Relaxation of convergence. This can be accomplished by a combined treatment with plus spheres, together with base-in prism. Incidentally, this type of training can be used with successful results in many types of asthenopia, such as the accommodative, ciliary spasm, or accommodation-convergence. The nervous, neurasthenic and emotional-hysterical types, tend to respond rather favorably to plus sphere and base-in orthoptic therapy. The lenses used may vary from +1.00 spheres to +2.50 spheres, depending on the age of the individual and the severity and extent of the defect.

The functional type of convergence insufficiency with ciliary spasm invariably requires base-out prism treatment. Plus lenses, when used, are most beneficial at the near point only. The plus lenses help to prevent over-stimulation of an already over-stimulated accommodation. Minus lenses are contra-indicated, because of their stimulative effect. The following two examples are cited to illustrate this point:

Mrs. G., secretary, age 42, complained of frontal headaches, severe eyestrain and inability to maintain work at the near point for any

length of time. Distant vision, while generally fairly good, has been of varying quality for several months prior to this consultation. This, by the way, coincided with a period of great mental and emotional stress. Spectacles which were prescribed about one year earlier seemed to help at first, but since the incidence of stress, have been ineffectual.

Visual acuity unaided or with glasses was 20/20-1 each eye. A change in the lens prescription was made which improved vision to 20/15 each eye. This also helped in near vision. Despite this, the symptoms and complaints of asthenopia persisted. The orthoptic examination indicated nothing abnormal in the motor or sensory functions. The cover test showed orthophoria. The convergence near point was 7 cm. without muscle deviation. The phorias were: P. R. 3Δ esophoria; P. P. 7Δ exophoria. The accommodation was subnormal. The amplitude was under 3 diopters each eye. All that was required to help her read comfortably was O. U. +0.50 D.S. over the distance correction.* Anything over that amount tended to blur vision at near. Fusion was normal, of low grade and maintained with effort. The visual fields were normal.

Following the first orthoptic treatment, lasting 20 minutes and consisting of base-in calisthenics on the Keystone teleye trainer, #2 rotor, with the Keystone base-in, "arrow" calisthenic cards, and O. U. +2.50 spheres in the instrument, Mrs. G. reported complete comfort. The headaches, which occurred daily, disappeared for several days, despite a heavy office schedule. A recurrent emotional upset brought back the asthenopia, but the symptoms were not as severe as formerly.

Mr. T., age 38, had complaints similar to those in the preceding illustration. In addition, he reported severe photophobia, tightness around the cranium, and pain in the eyes. He had no spectacles, and none were needed for either near or distance.

The orthoptic tests showed an accommodation amplitude O. D. = 8.50 D.; O. S. = 8.00 D. The convergence near point was 12 cm. The cover test showed no deviation. Ocular motility was normal. The phorias: P. R. 2Δ esophoria; P. P. 13Δ exophoria. No vertical motor defect. The ductions showed sub-normal adductive movements, while the abduction was within normal range. The positive and negative fusional reserves were erratic. Fusion was defective. The visual fields were essentially normal. The medical history listed emotionalism and neurasthenia, but in the opinion of his physician, the ocular complaints were a separate and distinct anomaly.

*O.U. +0.50⌒−0.50 D.C. × 180°.

Base-in calisthenics and plus spheres were not tolerated. However, when base-out calisthenics, using the Keystone equipment, were substituted (base-out arrow targets were used at the near point with O. U. +1.50 sphere), the response was excellent and symptoms disappeared.

Invariably, fusion must be considered in treatment. At times, if lenses are used, they are left in the cells of the instrument: at other times they are removed. The reason for this variableness is to test and determine reaction to treatment and its retentiveness.

A basic principle in the treatment of asthenopia, irrespective of type or causative factor, is *relaxation of accommodation*. The convergence factor is another matter. Treatment of convergence abnormality depends upon the factors producing such a defect. Simulated myopia produced by ciliary spasm requires proper differentiation and diagnosis. Similarly, convergence insufficiency produced by defective adductive movements must be differentiated from convergence weakness due to exhaustion of convergence produced by spasm of accommodation and the accommodative-convergence reserves.

Just as the diagnosis is different, so is the treatment different. Where differential diagnosis is inconclusive, trial and error period of treatment procedure may be advisable, before deciding on a definite routine. Once decided, such a routine should be followed to completion. In some cases of asthenopia, treatment results, though usually successful when persisted, may show slow response at the beginning. This is especially so in functional disturbances involving accommodation and convergence. Asthenopia resulting from under-corrected refractive errors tends to respond well and quickly to proper ophthalmic correction and orthoptics. The same holds for the aniseikonic, presbyopic, retinal, aphakic, astigmatic and anisometropic. Asthenopia in which the oculomotor system is a major factor, requires a broader treatment approach. It includes, besides eliminating the purely asthenopic symptoms, also elimination of the contributing factors. Oftentimes if the clinical findings so indicate, a complete system of orthoptics, for a specific motor defect must be included. Neurasthenic asthenopia, retinal, emotional, hysterical and related types, respond rather well to the relaxation of accommodation-convergence. Reflex, irritans, tarsal dolens and integrative asthenopia respond to removal of cause and to relaxation of the two primary functions of the visual apparatus—the accommodation-convergence.

SUMMARY

Asthenopia is not a specific refractive anomaly or pathological condition, but an aggregate of symptoms of disturbed physiologic functions. Many of these do not show either objectively or subjectively, but are

gleaned from the case history. There are several kinds of asthenopia, each with its own characteristic array of symptoms. Happily for the asthenopic patient this condition lends itself to correction. Recurrence is possible, if the original causes are reactivated. In this, asthenopia differs in no way from many other systemic and neurological disturbances.

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ANNOUNCEMENT

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NOTES ON THE OBJECTIVE DETERMINATION OF VISUAL ACUITY THROUGH OPTOKINETIC NYSTAGMUS*

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INTRODUCTION

Optokinetic nystagmus is physiological in nature and is commonly known as Railroad Nystagmus. It may be elicited from early infancy for the duration of the life span. Previously it was thought that the nystagmus could only be induced at the minimum age of 3 months but in 1957 Gorman *et al.*[1] induced the nystagmus on babies of 1-5 days of age. A 16 foot long piece of paper, 9 inches wide, running over two rollers was held tentlike over the head of the babies. Although the nystagmoid movements were very hard to see they were present. Gorman believed that at that age the vision was 20/670. This was based on the size of the characters and the distance at which it was seen to create the eye movements.

Optokinetic nystagmus can also be induced in animals, and many observations have been made on these. Both rods and cones play a part in the inducement of the nystagmus, and they form the basis for the determination of the visual acuity. Nystagmus can also be created through a pendulum arrangement, which was utilized by Goldmann.[2]

In the nystagmoid movements we differentiate between the gliding slow movement, often referred to as the leading movement, and called the slow phase of the nystagmus. Jerktype quick movements back to the point of origin are the rapid phase. The latter belong to the fixation movements. If an object falls on the periphery of the retina the fovea will immediately be directed to it. This fixation movement too is of the jerk type and involuntary. Kestenbaum[3] calls it the Einstellbewegung, an arbitrary turning in movement. Herring[4] saw in it a reflex occurrence in which the attention to the retinal image caused the reflex sensation, and both are called psycho-optical reflexes. They differentiate from the true unconscious reflexes through the cooperation of the attention and a longer latent stage.

The slow phase of the optokinetic nystagmus starts after a short

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latent interval of 0.25 sec. This depends on the speed of the movement of the visible object. If the speed of the object is too great, then the speed of the slow phase lags behind. A certain self regulatory mechanism will not allow the speed of the eyes to be greater than that of the speed of the visible objects. The carrier of the gliding movement is the fovea, and therefore we have a close connection with the fixation mechanism. According to Roelofs[5] the slow phase is the primary of the optic nystagmus, and the fast phase is only a succession of the long phase. The slow phase goes without latency over into the rapid phase; it is a saccadic movement of short duration of around 1/10 second, while the long phase is about again as long. Kestenbaum[3] saw in the rapid phase the return to the visual direction.

Attention increases optokinetic nystagmus. Accommodation can create a reduction but the amplitude becomes smaller. This must be taken in account when the working distance is short. Optokinetic nystagmus does not depend on illumination, provided the eye is adapted to the existing level. Only in great differences between light intensities are faulty results noticeable.

The size of the pupil has a certain influence on the visual acuity obtained objectively. Guenther[2] states that there is no strict correlation between visual acuity and pupil size. He admits however, that some subjects with large pupils may have a slightly higher acuity. The pupil size has to be recorded, and perhaps a line reduced from 20/20 to 20/25. Tiredness slows optokinetic nystagmus; therefore the examination should be done as quickly as possible.

As stated before, optokinetic nystagmus is a function of the whole retina. There is, however, difference of opinion whether the fovea or the periphery is the most important factor for the inducement of the nystagmoid movements. While central fixation is not absolutely necessary, the most important thing is the fixation *per se*. Cords[6] and others gave the fovea a greater intensity of the nystagmus, while Rieken[2] claims the greater value of the periphery as against the fovea, provided the eye was dark adapted. He further states that the periphery is in the recognition of rhythmic innervations better than the central field.

FREQUENCY AND AMPLITUDE

The oscillation frequency of the amplitude of the optokinetic nystagmus is the number of eye movements per second, and is best expressed in Hertz and depends on the rotating time. The number of Hertz is equal to the number of sensitized points of regard passing the center. The amplitude is the height of the movements measured in millimeters. Ohm[7] called the product of frequency, times the ampli-

tude, the energy of the nystagmus. The better the fixation the greater is the amplitude, and the amplitudes of both phases are equal, while the frequency is opposite to the amplitude and proportional to the speed of the object:

$$F = \frac{\text{speed}}{\text{amplitude}}$$

The index of the nystagmus is according to Ohm:[7]

$$\frac{\text{Energy of the right jerks}}{\text{Energy of left jerks}}$$

The product of this equation remains pretty constant even by changing the direction of gaze as Ohm[7] and Guenther[2] pointed out.

The speed of the moveable object of regard is best expressed by the angular velocity, as for instance the speed of the band passing through the frontal plane over the two rollers as in the instrument the writer has employed. It is best expressed in the Cords[6] formula

$$\frac{\text{Velocity}}{\text{sec.}} = \frac{\text{Length of band over the rollers}}{\text{Rotation time x Viewing distance.}}$$

However, a slower speed gives a greater innervation for the nystagmus but much depends on the apparatus. Ohm expresses himself in his writings for his instrument in angular speeds, while Guenther and others feel it is more exact to give the oscillation frequency in Hertz, the amount of sensitized objects passing one place of the retina in a certain interval. On the average this number of Hertz is 8 per second by equal intervals. Ohm[7] found very little difference in the nystagmus of 4-32 Hertz and says that even 1 Hertz could create almost normal optokinetic nystagmus.

The angular velocity is often given by the rotating time of the drum. This is not exactly correct as it is in relation to the viewing distance, that is the visual angle. To use Ohms figures as relating to his drum which is seen under an angle of 33 degrees and if the rotation time of the drum is 4 seconds, then the point of fixation travels from left to right or vice versa in 1.64 seconds over 33 degrees. Assuming the eye to be in a state of rest, the velocity is then $\frac{33}{164} = 20$ degrees per second. This is important as no nystagmus will be induced should this figure be higher or lower.

The visual angle is equal to the relative opening of the instrument. Under Ohm's arrangement of the rotating drum a relative opening of

3 to 5 degrees is needed. In some cases an opening of 1 to 1.5 degrees led to the release of nystagmus, which is rather fine and weak. Ohm used the method of Hebelnystagmography, and showed with accuracy the movements.

The innervation time is the duration under which the point of regard is seen, and lies between the first visibility on the one side until its ultimate disappearance on the other side. Diaphragms will of course reduce the innervation time. The movement from left to right is easier to induce and the nystagmoid movements are stronger than from right to left.

I should call attention to after nystagmus. The optokinetic after nystagmus is that movement that still exists after the innervating objects have passed the innervated field. The eyes do not come to an abrupt halt, but rather show a continuation of rhythmic movements in the direction of the nystagmus but less strongly.

Attention should be paid to disturbances of the optokinetic nystagmus, as it is usually due to pathology. Guenther[2] classified them:

1. Complete absence or lessening of the nystagmoid movements to both sides.

2. Absence or lessening of the nystagmus to one side, while the other shows normal optokinetic movements.

INVERSION

Inversion is a reversal of the movements. Duke-Elder states[8] that inverse optokinetic nystagmus occurs where the more rapid movement is in the direction of the moving object. Inversion may occur in congenital or amblyopic nystagmus of ocular origin. It is often associated with true nystagmus, which takes precedence over the optokinetic type. In the slow type the rapid phase is slowed due to the movement of the opposite direction, and the rapid phase becomes actually the slower one. In the rapid type the fixation can not be maintained because of the congenital nystagmus. Due to quick and sharp movements the rapid phase seems to be accelerated.

Latent nystagmus is seen rather seldom, and never binocularly but will appear when one eye is occluded, and those eyes are not normal.

Amblyopic nystagmus is to be considered, as we might try to obtain the visual acuity by optokinetic nystagmus. It is essentially a fixation nystagmus, due to poor central vision. Unilateral amblyopia is seldom the cause for nystagmus. Color blindness on the other hand is.

Pendular nystagmus is of pathological origin. These are usually eyes with very poorly developed optomotor reflexes, and the eyes are hardly ever still.

Optokinetic nystagmus can not be used for the determination of the resolving power of the eye in subjects with:

1. Existing large nystagmus.
2. Disturbance of the central nervous system.
3. Unability to observe.

In existing nystagmus it is obvious that the stronger movements would overshadow the optokinetic one.

THE GOLDMANN EYE PENDULUM

The procedure of the examination under the Goldmann principle was described in a previous paper.[9] It actually is an optokinetic reaction but not of the same type as the optokinetic nystagmus. The latter is induced by the continuous movement of the innervating object which the eye follows, and is based on the slope of the sensitivity of the retina, while the eye pendulum does not have the eye to follow its innervating object. Rather it is fixed with the fovea on the swinging test object, and is led so to both sides. Through the diminishing of the resolving power of the eye towards the periphery all objects appear gray and stationary. The eye pendulum can be created through various arrangements, and according to the swing larger or smaller synchronic eye movements are noticeable, and are seen on the kryptomograph as sinus curves.

Using the Goldmann method of the pendulum, the writer can not see its usefulness in malingerers nor patients of low I.Q. as it demands perfect fixation of the target or grid. It might well be the best objective method of measurement of the foveal acuity. The instrumentation is complex, and the exactness demanded thereof makes it impractical. Another factor is that low visual acuities, under 20/80 for instance, can not be measured.

The basis for testing visual acuity objectively is optokinetic nystagmus in its broadest sense. Ohm[7] used the stoppage of the eye movements through a sensitizing object consisting of broad stripes. Goldmann used optokinetic sensation through a pendulum. In the first case we have the eye movements coming to a halt, and in the second the fixating and quiet eye will show the eye pendulum, just the opposite of Ohm. In each case the change represents the threshold, and gives the visual acuity for the prevailing test object.

The use of optokinetic nystagmus for the objective determination of visual acuity is further helped since the kinetic sensitivity of the retina becomes less when we move from the fovea to the periphery. Even highly defective eyes react to the nystagmus. Of course we have to find the proper sensitizing size object and its optokinetic sensitivity.

THE GUENTHER METHOD

Guenther's method[2] has the advantage that it has more constants than the others. We have a constant size of the innervated field as a change of the size of the field would result in changes of the amplitude of the nystagmus. The principles of his method resolve themselves into 5 factors:

1. Size of innervated field.
2. Innervated object, the two together he calls the test object.
3. Distance.
4. Speed of object moving through the visual angle.
5. Number of Hertz.

In order to induce optokinetic nystagmus, we must have a test object in which we differentiate between the stimulated field and the stimulated objects.

The size of the innervated field is constant between 2 to 3 degrees in contrast to Ohm's 33 degree horizontally. The constancy of the visual field at the various distances is assured by changeable diaphragms, 5, 10 and 15 cm. squares. Only in special cases such as a central scotoma larger than 3 degrees does Guenther use a larger innervated field up to 17 degrees. Even with a scotomata as large as 15 degrees the visual acuity can be easily determined with reasonable accuracy. Since the innervating field is so small it has its own character, and as we deal with optokinetic nytagmus created by this small field, and since this brings about a very small amplitude, we need the magnification of a loupe to see the movements exactly. Guenther points out that theoretically we should not call the movements nystagmus and he referred to them as optokinetic eye movements, and of these only the rapid phase can be recognized.

To find the smallest sensitizing field that still created the optokinetic nystagmus, he used a 25 mm. diaphragm with test objects of the checkerboard type, 5 and 2 mm. respectively. He used persons with 20/20 visual acuity or better, and after testing 10 persons the average size of the sensitized field 25′ to 30′ with no optokinetic reaction; 32′ the first jerks; 40′ clearer visible jerks, and 48′ to 60′ clear optokinetic reaction.

The stimulating objects consist of black and white chessboard targets 10, 5 and 2 mm. squares. Through variation of the distances from 0.5 to 4.5 meters, we receive visual angle values from 1.5 minutes to 60 minutes. With an optokinetic minimum of 2′ angle, we will have visual acuities ranging from 20/600 to 20/15.

The instrument consists of a black box without sides and backwall. In the center is a window for the exchangeable diaphragms of 5, 10 and 15 cm. squares. In back of the window are 2 cylinders of 9.5 cm. in diameter over which runs the 100 cm. ribbon with the black and white chessboard targets of 2, 5 and 10 mm. respectively. The ribbon runs from right to left or vice versa, and 2 40-watt lumiline bulbs illuminate the field evenly. A motor rotates the cylinders.

An observation viewing device, 20 cm. in length and 3.5 cm. in diameter is needed, which is adjusted to the eye to be examined: for further reducing the sensitizing field a diaphragm is at the end of the tube facing the instrument. A chin rest serves to steady the head and aid in the observation. When the tube is adjusted to the eye a small image on the cornea is visible: this is particularly important for the examination of malingerers. To be able to see the optokinetic movements a 13 D. loupe and a small flashlight are needed. The beam is directed to the temple of the eye under examination: small but strong reflex images, whose rhythmic movement tells of the appearance of the optokinetic nystagmus, are observed.

The examination is made in a semidarkened room, and the patient has to be in the room for about 5 to 10 minutes to adapt to the prevailing illumination. There is no other light in the room except that of the instrument. The examination is monocular and the other eye is completely occluded.

Guenther starts with values below the threshold and increases them gradually. It is best to start with ½ of the actual acuity. Of further importance is the choosing of the field size of the checker board grid.

Annoying eye movements do often hinder the observation of the nystagmoid movements. Guenther classified them as arbitrary and non-arbitrary eye movements. To the non-arbitrary ones belong the physiological eye unrest or pseudo-nystagmus. The opening and closing of the eyelids can also be a source of annoyance. In most cases the optokinetic excitation subdues the existing eye movements, while a speedup of the examination will overcome the arbitrary eye movements: an attempt should be made to arouse a greater interest in the patient.

RESULTS

The creation and reading of the nystagmoid movements demands a certain experience to ascertain the movements correctly, as from this observation depends the exactness of the visual acuity. This is especially important where preexisting nystagmus is present or the before mentioned arbitrary and non-arbitrary eye movements tend to influence the examination.

Special attention must be called to those subjects with low visual acuity, 20/80 or lower. Here often the objective visual acuity is higher than the subjective. In Guenthers instrument this can be overcome by:

1. A better Hertz amount using object size 10 mm.
2. Relative larger innervated field.
3. The higher contrast in the checker board test objects.

It is the writer's opinion that the latter is responsible for my findings as well as Guenther's to be better than those of Reinecke.[10] Reinecke used an instrument designed by Gorman of Boston, which was similar in design to mine except for the test objects. Its test objects consisted of vertical stripes moved horizontally before the eyes, and the lines were of diminishing thickness. The examination procedure was the same as the writer's, except for the distance which was from 1 to 2.5 meters. He examined 26 subjects and found good correspondence in the lower visual acuity, while the higher visual acuity did not correspond with each other.

My tests and those of Guenther showed that in the better visual acuities we had a close relationship. When the patient was fully corrected and placed before the instrument the values obtained objectively corresponded closely to the ones obtained subjectively, and this is true in all refractive anomalies, including aphakia. In amblyopia the objective visual acuity as a rule is lower, especially in convergent squint. Here the subjective visual acuity seems to be better. Even in those cases I would advocate a test before the nystagmoid drum. Developing cataracts or cloudy media showed a difference too, and the same is applicable in other pathology associated with lower visual acuities.

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AMERICAN ACADEMY OF OPTOMETRY

NEW MEMBERS

The following optometrists and educators have been elected Fellows of the American Academy of Optometry. The Executive Council welcomes these new members.

Mr. Eric B. Bell, 30 Albert Street, Mansfield, Notts, England.

Dr. Stanley Eisenberg, Powell Professional Bldg., 2234 Jackson Avenue, Seaford, New York.

Dr. Robert C. Garvin, Jr., 3321 N.W. 17th Avenue, Miami, Florida.

Lt. Martin Gellman, United States Naval Hospital, Great Lakes, Illinois.

Dr. Robert Greenberg, 104 West Jefferson Street, Tallahassee, Florida.

Mr. Maxwell M. Lang, Department of Optometry, The University of New South Wales, Broadway, Sydney, N.S.W., Australia.

Dr. Roger O. Lindquist, 612 West Broadway, Minneapolis, Minnesota.

Dr. Leo J. Leydon, 219 Railway Avenue, Weyburn, Sask., Canada.

Dr. Carl R. Muth, 174 West Line St., Bishop, California.

Dr. Herbert S. Player, 306 Regent Court, Stockton, California.

Mr. Jurg von Guten, Bundesgasse, Bern, Switzerland.

AMERICAN JOURNAL OF OPTOMETRY
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THE ACADEMY SECTIONS

Members of the Academy who regularly attend the annual meetings are quite familiar with the five Academy Sections and how they operate. There are members, however, who have not attended these annual conventions and at times these seem puzzled as to how a member of the Academy gets to belong to a Section: what it costs: how they are organized: what it is they are expected to accomplish and how they function.

Any member of the Academy may join one or more Sections of his choice. There are no dues or fees or special assessments and all it requires is that the member attend those Section meetings of interest to him during the annual convention. There he will become acquainted with optometrists who have similar interests and also with those members serving on the committee that supervises the activities of the Section.

The Sections are organized very simply. Each year, during the

annual convention, the President of the Academy visits and consults with each of the Sections and gets from these groups suggestions for the committee appointments for the following year. These suggestions are later placed before the Executive Council and, following approval, become the new Section committee. In this way the Sections are to a large degree responsible for their own management.

Now what is expected of a Section? Its duties are to review all current progress in its field and to keep Academy members aware of new developments, successes and problems. The chairman of each Section serves on the Editorial Council of the Academy Journal and has the final vote of approval or rejection on doubtful or questionable papers submitted for publication that fall within the scientific and clinical scope of the Section. The Section developes its own program for its sessions during the annual convention.

Each Section meets on three occasions during the annual meeting of the Academy. Those in attendance hear papers largely dealing with the clinical aspects of the specialty and discuss questions or problems as these are presented by the chairman. Questions are decided in the usual democratic manner. The Executive Council of the Academy may be called in for help but only at the Section's request.

Much of the advancement made in the refractive and allied eye care fields during the past twenty years has been due to the month in and month out activity of the Academy Sections each working to further the devolopment of their particular specialties. The Academy and the entire profession is deeply grateful to those Section leaders who over the years have done so much to enlarge our knowledge and skills. Academy Section committees continue their work throughout the year by correspondence and at local Chapter meetings.

A list of the present Section committees and the papers and subjects that will be presented at the coming Section meetings December 10-12, 1961, at the Drake Hotel, Chicago, Illinois, follows. All Academy members and their guests are urged to take an active part in one or more of these five fine programs.

CAREL C. KOCH

TRANSACTIONS OF THE AMERICAN ACADEMY OF OPTOMETRY

A department devoted to announcements, reports, appointments, organization data, news, professional problems and ideals, as these relate to the Academy.

SECTION PROGRAMS

AMERICAN ACADEMY OF OPTOMETRY

Drake Hotel

Chicago, Illinois

Section Meetings, December 10, 11 and 12, 1961

SECTION ON CONTACT LENSES AND SUBNORMAL VISION AIDS

SUNDAY, DECEMBER 10, 1961. 8:00 A. M.

Lester A. Dine. Contact Lens Photography.

David Huang, O.D., Clinical Experience in Contact Lens Fitting.

Donald R. Korb, O.D., The Polariod Photographic Technique of Fittting Contact Lenses.

Philip R. Haynes, O.D., Some Optical Considerations in Keratopographometry.

William Sohnges, Recent Contact Lens Development in Germany.

Charles A. Bayshore, O.D., Apical Clearance Fitting of Small Ventilated Contact Lenses.

Leslie Evershed-Martin. The Value of Keratometric Statistics.

MONDAY, DECEMBER 11, 1961. 8:00 A. M.

Tom H. Brungardt, O.D., Study of Corneal Contour Measurements.

Allen A. Isen, O.D., Prism Ballast Lenses.

William Feinbloom, O.D., Ph.D., An Ellipsoidal Surfaced Contact Lens.

Newton K. Wesley, O.D., Fitting the Corneal Contour.

Harold I. Moss, O.D., The Lens-Cornea Relationship.

Henry A. Knoll, Ph.D., Corneal Topography as Revealed by the Photokeratoscope.

Arnold L. Langsen, O.D., Criteria for Completing a Successful Case.

TUESDAY, DECEMBER 12, 1961. 8:00 A. M.

A. E. Reynolds, O.D., The Present State of Contact Lens Science.

Bradford W. Wild, Ph.D., and Richard L. Shroder, O.D., Variability of Base Curve in Contact Lenses.

John R. Kennedy, O.D., Corneal Lenses in Active Pathology.

George N. Jessen, O.D., Newest Developments in Contact Lenses.

I. Irving Vics, O.D., Physiologic Eye Changes Under Hypnosis.

Daniel O. Elliott, O.D., The Manufacturing of Contact Lenses.

Allen A. Isen, O.D., Clinical Teaching Systems for Contact Lens Courses.

SECTION ON CONTACT LENSES AND SUBNORMAL VISION AIDS: Dr. Robert C. Graham, Pasadena, chairman: Dr. Daniel O. Elliott, Jr., South Bend, Indiana, vice-chairman: Dr. Bernard Mazow, Coral Gables, Florida: Dr. John C. Neill, Philadelphia: Dr. William W. Policoff, Wilkes-Barre, Pennsylvania: Dr. Harold Moss, Woodbridge, New Jersey: Dr. Otto J. Bebber, Lakewood, Colorado.

SECTION ON BINOCULAR VISION AND PERCEPTION

SUNDAY, DECEMBER 10, 1961. 9:00 A. M.

Bernard E. Vodnoy, O.D., Latest Improvements in the Aperture, Rule and Variable Prismatic Mirror Stereoscopes in Orthoptics.

Bradford W. Wild, O.D., Ph.D., Some Visual Correlates of Eye Movements.

Harold S. Soland O.D., Reading Improvement Training in Office Practice.

MONDAY, DECEMBER 11, 1961. 9:00 A. M.

William R. Baldwin, O.D., M.S., Historical Development and Review of the Literature in Pleoptics.

Ellen S. Takahashi, O.D., Demonstration and Evaluation of Pleoptic Instruments.

William Smith, O.D., D.O.S., Variations in Concepts of Pleoptic methods and Instruments.

TUESDAY, DECEMBER 12, 1961. 9:00 A. M.

Bernard E. Vodnoy, O.D., The Utilization of Haidingers Brushes in Orthoptics.

Donald R. Korb, O.D., A Comparative Analysis of the Effects of Spectacle Lenses and Contact lenses on Binocular Vision.

Raymond R. Roy, O.D., Prism and Binocular Stress.

SECTION ON BINOCULAR VISION AND PERCEPTION: Dr. Max Schapero, Los Angeles, chairman; Dr. Ernest Giglio, Bernardsville, New Jersey; Dr. Merton C. Flom, Berkeley; Dr. Ralph LeDrew, Edmonton, Alberta, Canada; Dr. Darrell B. Carter, Houston; Captain Donald G. Pitts, Wright-Patterson Air Force Base, Ohio; Dr. Bradford W. Wilde, Columbus.

SECTION ON PATHOLOGY

SUNDAY, DECEMBER 10, 1961. 9:00 A. M.

Frederick W. Hebbard, O.D., Ph.D., Pressure and Flattening in Tonometry.

Bernard E. Vodnoy, O.D., Home Screening of the Visual Fields.

Carel C. Koch, O.D., Interprofessional Relations.

Panel Discussion.

MONDAY, DECEMBER 11, 1961. 9:00 A. M.

Arthur Shlaifer, O.D., Ph.D., A Review of the Recent Literature in Glaucoma.

Frederick W. Hebbard, O.D., Ph.D., Clinical Experience with the Mackay-Marg Electronic Tonometer.

Panel Discussion.

TUESDAY, DECEMBER 12, 1961. 9:00 A. M.

David Simon, O.D., Optometric Use of the Slit Lamp.

Harold Simmerman, O.D., An Illustrated Review of Macular Degeneration.

SECTION ON PATHOLOGY: Dr. Arthur Shlaifer, Philadelphia, chairman; Dr. Walter W. Perrott, Jr., Columbus, Georgia; Dr. Harold Simmerman, Wenonah, New Jersey; Dr. Monroe J. Hirsch, Ojai, California; Dr. Stephen C. Outlaw, Albany, Georgia; Dr. Robert J. Johnson, Latrobe, Pennsylvania; Dr. Ralph B. Gaeta, Ramsey, New Jersey; Dr. Hernando Henao, Jr., Colombia, South America.

SECTION ON REFRACTIVE PROBLEMS AND ANISEIKONIA

SUNDAY, DECEMBER 10, 1961. 9:00 A. M.

John Davis and Arline Rayner. Lens Design in Aniseikonia.

Panel Discussion.

MONDAY, DECEMBER 11, 1961. 9:00 A. M.

Arline W. Rayner. Use of Simplified Tables in the Construction of Bitoric Lenses.

Panel Discussion.

TUESDAY, DECEMBER 12, 1961. 9:00 A. M.

Elwood H. Kolb, O.D., The Examination of the Retarded Child.

Robert Bannon, O.D., D.O.S., New Instrumentation.

Otto R. Engelmann, O.D., Clinical Demonstration of the Ophthalmodiastimeter Technique as used in Refraction, Orthoptics and Bifocal Fitting.

Panel Discussion.

SECTION ON REFRACTIVE PROBLEMS AND ANISEIKONIA: Dr. William P. Schumann, Vineland, New Jersey, chairman; Dr. Oscar L. McCulloch, Holyoke, Massachusetts; Dr. Robert E. Bannon, Buffalo; Dr. Edwin W. Bechtold, Middle Village, New York; Dr. Leo F. Madigan, Boston; Dr. Harold M. Fisher, New York City; Dr. Robert J. Davis, Henderson, Kentucky; Dr. Arene T. Wray, Los Angeles.

SECTION ON PUBLIC HEALTH AND OCCUPATIONAL VISION

SUNDAY, DECEMBER 10, 1961. 9:00 A. M.

Felix A. Koetting, O.D., Optometric Participation in Out-Patient Clinics and Criteria for Maintenance of Standards.

Panel Discussion.

MONDAY, DECEMBER 11, 1961. 9:00 A. M.

Clark W. Mangum, Jr., M.D., Optometry's Participation in Community Health Service.

Panel Discussion.

TUESDAY, DECEMBER 12, 1961. 9:00 A. M.

I. W. Silberstein, O.D., Contact Lenses in Industry.

Felix A. Koetting, O.D., and Robert C. Mueller, O.D., Evaluation of a Rapid Stereopsis Test.

SECTION ON PUBLIC HEALTH AND OCCUPATIONAL VISION: Dr. Gayland F. Kintner, Lynden, Washington, chairman; Dr. George C. Hill, Portsmouth, Virginia; Dr. Herman Sager, Lake Success, New York; Dr. Richard Feinberg, Washington, D. C.; Dr. Sylvester K. Guth, Cleveland; Dr. Henry W. Hofstetter, Bloomington, Indiana; Dr. Chester H. Pheiffer, Houston; Dr. Lois B. Bing, Shaker Heights, Ohio; Dr. Donald D. Slater, Hot Springs, South Dakota.

All of these Section Programs are subject to some final revision. The General Program of the annual convention of the American Academy of Optometry, December 9-12, 1961, the Drake Hotel, Chicago, Illinois, was published in the October issue of this journal. The now slightly revised General Program and the individual Programs of each of the Sections will be distributed at the Registration Desk during the convention.

NOTES ON PROGRAM OF THE CONTACT LENS SECTION

Because of the wide spread interest in contact lens fitting and because of the quality of the Program arranged for the Academy Contact Lens Section, December 10-12, 1961, at the Drake Hotel, Chicago, we are submitting a series of short reviews of some of the many papers now listed on the Section Program in this issue. These reviews are presented by Dr. Robert Graham, chairman of the Section.

Lester A. Dine, Contact Lens Photography. The single lens reflex camera: its use and models available. The black light equipment, showing how same is mounted on the camera, and how the light revolves around the lens. Procedure in taking pictures. Slides showing routine pictures.

David Huang, O.D., Clinical Experience in Contact Lens Fitting. This report attempts to help the beginning contact lens practitioners save time and error in patient control and lens selection. A detailed analysis of one hundred clinical cases will be presented including the description of a general fitting procedure, wearing time schedule and a review of patient indoctrination and doctor-patient relationship.

Phillip R. Haynes, O.D., Some Optical Considerations in Keratopographometry. The paper will discuss various optical problems associated with measuring the topography of the cornea. Comments will be made on instrument design and accuracy.

Charles A. Bayshore, O.D., Apical Clearance Fitting of Small, Ventilated Contact Lenses. Presenting the changes in technique and the results after more than two hundred cases have been fitted with lenses of this type. This fitting was used mainly in hyperopic cases, occasionally in problem cases.

Tom F. Brungardt, O.D., Study of Corneal Contour Measurements. Corneal measurements were collected from two sources: one clinical, the other photographic. These data were treated statistically for correlations and predictive values. Measured values were central Keratometric reading, corneal diameter and flattest peripheral curve. Related measurements, not clinically available, are discussed as they pertain to contact lens fittings.

Allan A. Isen, O.D., Prism Ballast Lenses. Experience with fitting prism ballast lenses both round and out of round (½ truncated, double truncated, etc.). There are a number of variables involved in these lenses, horizontal diameter, vertical diameter, amount of prism, curvature of the top edge, etc.

William Feinbloom, Ph.D. An Ellipsoidal Surfaced Contact Lens. Starting with Helmholtz's data showing the cornea as an ellipsoid, a

method for making ellipsoidal surfaced contact lenses to fit this data is described. A clinical method of obtaining the data necessary to fit the proper equations in order to calculate the form of ellipse for an individual eye is presented. The clinic results in 100 cases fitted with ellipsoidal surfaced contact lenses are included. Demonstrations and detailed discussion.

Harold I. Moss, O.D., The Lens-Cornea Relationship. The paper will be based upon a photographic analysis of lenses on the same cornea. All lenses will have the same base and peripheral curves as well as overall diameter. The only variation will be the optic zone diameters of the lense. The evidence will clearly indicate that one must provide for the corneal variables when prescribing or will, within time, run into corneal insult and/or exhaustion.

Arnold L. Langsen, O.D., Criteria for Completing a Successful Case. There has been a plethora of articles in recent years concerning methods for objectively determining the correctness of the relationship between a contact lens and the cornea. The great number of points of view presented, many of them diametrically opposed, has lead to an almost chaotic state of confusion in the contact lens field. The great disparity of recommendations for lens design and fitting procedure, and the claim for success with all of these methods, suggests to the author an evaluation which will lead to criteria, common to all methods, for successfully completing a contact lens case.

Bradford W. Wild, Ph.D., and Richard L. Shroder, O.D., Variability of Base Curve in Contact Lenses. Contact lenses that had been cut from rod stock were subjected to various environmental conditions. It was found that the radius of the base curve of a contact lens tends to lengthen when the lens is soaked in water at a temperature of 75° F. for five days. If the lens is allowed to dry, it tends to revert to its original base curve, although there are many exceptions to this generalization. If a lens is soaked in water at 95° F. for five days the base curve radius tends to lengthen to the same extent as that found at the lower temperature. Statistical treatment of the data shows low correlations between the change in base curve and the dioptric power of the lens, as well as between the change in base curve and the original radius of the base curve, and between the change in base curve and center thickness.

John R. Kennedy, O.D., Contact Lenses in Active Corneal Pathology. A discussion of the use of contact lenses on a patient with iritis and corneal dystrophy; on an aphakic patient with intermittent glaucoma; on a patient with phthisis and uveitis as a result of sympathetic ophthalmia.

I. Irving Vics, O.D., Physiologic Eye Changes under Hypnosis. The literature to date has established that "All the physiologic variables that have been investigated in the hypnotic state under basal conditions, are found to be unchanged when compared to the waking condition" (Gorton). This pilot study is the first eye investigation under hypnosis in which measurable physiologic changes were discovered.

Daniel O. Elliott, Jr., O.D., The Manufacturing of Contact Lenses. This paper argues the proposition that the local production of contact lenses would be of service to the optometrist, the optician and the public. In order to accomplish this, an orderly and precise method of production of contact lenses, suitable for use by the optician is presented. The paper presents a discussion of the present obstacles to the accomplishment of local contact lens production and how they can be overcome. Plastics and plastic forming are also discussed as they relate to contact lens production.

CURRENT COMMENTS

Terry Judith Parkins
Editorial Assistant

Optometrists will confer a favor by sending news items of general interest for this department; such as relate to new instruments, clinical techniques, education, visual health and optometric legislation and organization.

FALL MEETING OF THE MICHIGAN CHAPTER

The Michigan Chapter held its Fall 1961 meeting at the Kellogg Center of Michigan State University. In keeping with the Chapter program of cultural lectures combined with optometric lectures, the morning program was presented by Dr. Harm J. deBliz. He spoke on "A Geographer Looks at South Africa." He had lived in Nigeria for many years where he was teaching at the University. He was forced to leave South Africa because of his personal opposition to the Apartheid Policy. Dr. deBliz discussed the discovery, the colonization, and the eventual fight for control of South Africa by the Dutch and British. He discussed how the native Negro Africans have been severely treated and forced to live under conditions worse than slavery. All the twenty-seven members who were present are looking forward to obtaining his new book FACTS AND FRICTION, which will be published in February. In it he describes the problems that exist in South Africa and the preservation of human dignity.

Dr. Bradford Wild gave the optometric lecture in the afternoon. He outlined the Orinda Study and gave the tests that must be performed

in school visual screening to give adequate information concerning the children's vision. He also discussed "The Effect on Contact Lens of Corneal Physiology." Dr. Wild discussed the changes that the cornea undergoes when a contact lens is worn. Based on these changes, a minimum basic fitting procedure was evolved.

DREW JOINS LIBERTY

Ralph Drew, author, lecturer and authority on ophthalmic frame styling, has joined Liberty Optical as director of professional relations. Mr. Drew is best known for his informative series of brochures—written exclusively for Liberty Optical—on *The Right Eyewear at The Right Time*, the technique of fitting fashion frames to the individual. Ever since he mastered the intricacies of dispensing, Mr. Drew has been serving the ophthalmic needs of the public, first in his native England and for the past 14 years in the United States. Until recently, Mr. Drew had been Director of Public Relations for Doig Optical Company, Pittsburgh, Pennsylvania.

PHIL SILVERS HEADS CENTER'S APPEAL

Mr. Phil Silvers, star of TV and the stage and currently appearing in the smash Broadway musical comedy, "Do Re Me," has accepted chairmanship of the Optometric Center's national appeal for its Indigent Visual Care Program. Mr. Silvers who is himself nearsighted indicated to the Center's Trustees that he accepted the chairmanship because he "recognized the forthright and humanitarian endeavors of the Optometric Center of New York to bring vision and hope to those whose lives lacked them."

"This institution, by herculean efforts for its size, has reached into the community with a helping hand and has provided free professional care to thousands of needy men, women and children. Yes, indeed, let us not forget the children who, in their formative years, must not be deprived of a blessing as cherished as any a human being could hold dear. The Center has given the very best that Optometry has had to offer and by that generosity it has earned the admiration and respect of the whole community. I appeal to all who read this message to help the Center and its devoted staff continue the vital work of vision care for the less fortunate. Send contributions to the Optometric Center of New York, 351 West 48th St., New York 36, New York."

Dr. Alden N. Haffner, executive director of the Optometric Center, has indicated that this national appeal to all optometrists in the United States by Phil Silvers will be conducted through the end of the year. The Fund Raising Chairman for this appeal is Mrs. Billie M. Lyons.

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Dr. Sheard was formerly editor of the *American Journal of Physiological Optics* and for many years director of the Department of Biophysics, The Mayo Foundation. The book contains 83 illustrations, 476 pages, $15.00.

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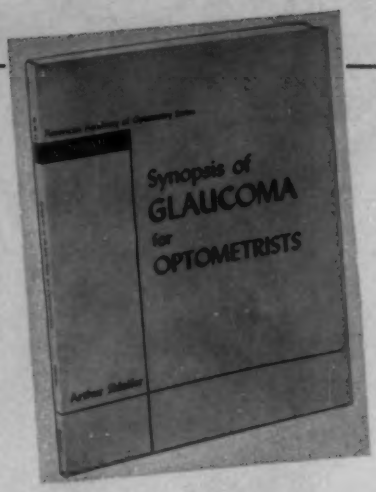

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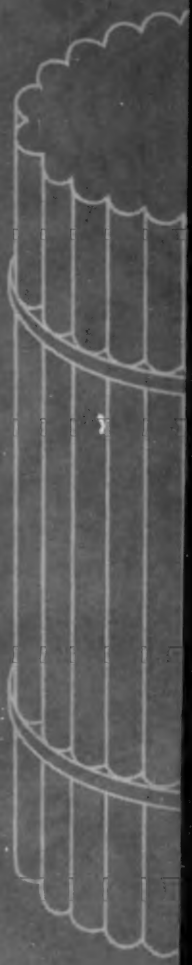

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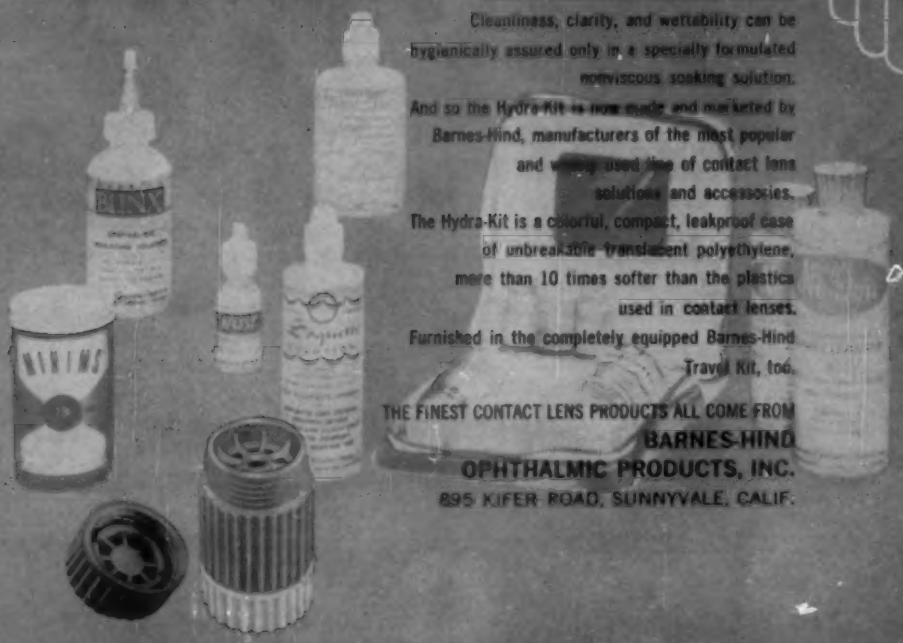

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